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CONTACT ULCER OF THE LARYNX

PATHOLOGIC OBSERVATIONS

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Contact ulcer is a particular type of chronic inflammatory laryngeal lesion localized at the vocal processes of the arytenoid cartilages and associated with typical clinical features and symptomatology. It was first described by Chevalier Jackson, who gave it the name by which it is now known. He recorded having observed two hundred and seventeen cases.[1] Other cases have been reported subsequently.[2] My own observation includes fifteen additional cases in all of which there were associated pathologic conditions of the nose and nasal accessory sinuses.

The name, contact ulcer, is particularly suitable because it gives a good conception of the laryngeal picture, the pathologic conditions, the etiology and the perpetuating factors of the disease on which its chronicity depends.

Of all the different types of localized chronic inflammation of the larynx, contact ulcer has the most distinct clinical characteristics. Localized manifestations of chronic laryngitis are not uncommon and may be in the nature of superficial erosions, but contact ulcer has a distinctive character and clinical course.

Contact ulcer may be described as a shallow ulcerative lesion localized on one or both sides of the larynx in the region of the vocal process of the arytenoid cartilage. During phonation, the ulcerative lesion, if unilateral, comes in contact with the vocal process of the opposite side, or if bilateral, with the opposite ulcer. Etiologically, the chief factor seems to be the mechanical one of pressure contact during phonation. Predisposing factors are the professions and occupations that are associated with vocal abuse of the larynx. My clinical studies lead me to believe that contact ulcer is not uncommonly associated with chronic inflammation of the nasal accessory sinuses.

From the Instituto di Anatomia Patologica, Université di Milano.

Read at the Congress of the Pan-American Medical Association, Dallas, Texas, March 21, 1933.

1. Jackson, Chevalier: Contact Ulcer of the Larynx, Ann. Otol., Rhin. & Laryng. **37**:227 (March) 1928.

2. Jackson, Chevalier, and Jackson, Chevalier Lawrence: Contact Ulcer of the Larynx, in Jackson and Coates: Nose, Throat and Ear and Their Diseases, Philadelphia, W. B. Saunders Company, 1930, p. 807.

In all of the cases reported by Chevalier Jackson in which a biopsy was made, a histologic picture typical of chronic inflammation and ulceration was present without any sign of malignant or tuberculous disease.

Symptomatologically, the disease is manifested in hoarseness, often slight, and pain or discomfort localized in the throat and usually referred by the patient to the region of the larynx itself; occasionally the pain radiates to the ears and may be severe.

The laryngoscopic appearances are those of an inflamed mucosa with ulceration involving the mucosa of the perpendicular surfaces of the opposing arytenoid eminences. In some instances the denuded vocal process of the ulcerated arytenoid cartilage is found in the bed of the ulcer. The ulcer usually surrounds this necrotic vocal process. Chevalier Jackson found that recovery occurred in all patients who followed strictly the instructions as to vocal rest; but he recorded the disease as always chronic in its course. In a few cases he found that cancer ultimately developed, and he has also observed cases of nonspecific contact ulcer of the larynx associated with tuberculosis of the lungs. In two of the latter cases the ulcer ultimately became tuberculous. The lesion is, however, distinctly an inflammatory one and bears no more relation to cancer or tuberculosis than does any other inflammatory laryngeal lesion.

It was my privilege to observe two cases from the pathologic point of view, typical contact ulcers being found incidental to autopsy in patients dying of diseases unrelated to the laryngeal lesion. One of the cases was discovered by my friend and collaborator, Dr. Enrico Bozzi, who has given me the case for description. The other case I observed myself. These two cases are interesting because of the histologic observations, which seem to be the first so far made from the pathologic point of view in which both the macroscopic and the histologic findings are available; these findings are in perfect accord with the clinical description as given by Chevalier Jackson.

REPORT OF CASES

CASE 1.—A man, aged 26, died of pulmonary tuberculosis. At autopsy extensive tuberculous lesions were found in both lungs with cavitation in both upper lobes and diffuse peribronchial tuberculous lesions in both lower lobes. There was a moderate degree of degeneration in the muscle of the heart and in the liver, but no other lesions and no tuberculosis of the intestine were apparent. It was noted particularly that there was no tuberculosis of the hypopharynx. The larynx was removed. The epiglottis and ventricular bands were normal. Grossly, the anterior two thirds of the vocal cords seemed normal, and no pathologic change was noted in the posterior commissure. In the region of the arytenoid process on each side of the larynx there was a symmetrical excavation about 3 mm. in length and about 2 mm. in depth; the edge of the ulcer was irregular. In the bed of the ulcer there emerged the vocal process of the corresponding arytenoid cartilage. No accumulated exudate was seen in either excavation.

Histologic examination of longitudinal sections of each vocal cord, including the ulcerated portion, showed no great degree of pathologic change on the two anterior thirds of the cord; there was a mild inflammatory infiltration around the mucosal

Fig. 1 (case 1).—Bilateral ulceration of the vocal cords posteriorly in the region of the vocal process. For clearness of photographic reproduction black cotton was inserted into the contact ulcer on each side (*A* and *B*).

Fig. 2 (case 2).—Bilateral ulceration of the vocal cords in the region of the respective vocal processes (*C* and *D*). In the bed of both ulcers the vocal process of the arytenoid was palpable with a probe.

glands in the subglottic region. On the posterior third near the arytenoid cartilage there was an excavation reaching into the cartilage itself, so that the vocal process lay bared in the cavity of the ulcer. The walls of the cavity were composed of

granulation tissue having the usual appearance of chronic inflammation but without any epithelial proliferation. The inflammatory wall was about 1 mm. in thickness and showed no tuberculous characteristics. A slight degree of inflammatory infiltration was noted between the musclar fibers at the periphery of the ulcer. The vocal process of the arytenoid cartilage which protruded in the bed of the ulcer showed superficial necrosis.

CASE 2.—A man, aged 52, died of cancer of the stomach. Autopsy revealed an adenoma of the thyroid, a carcinoma of the stomach, with metastatic invasion of the pleura, and diffuse bronchopneumonia of the inferior lobes of both lungs. Metastatic lesions were found in the spleen, kidney and liver. There was nothing in the history of the case so far as it was obtainable that would indicate laryngeal pathologic changes; the larynx was opened in the course of a routine postmortem examination. The endolaryngeal pathologic conditions were grossly similar to

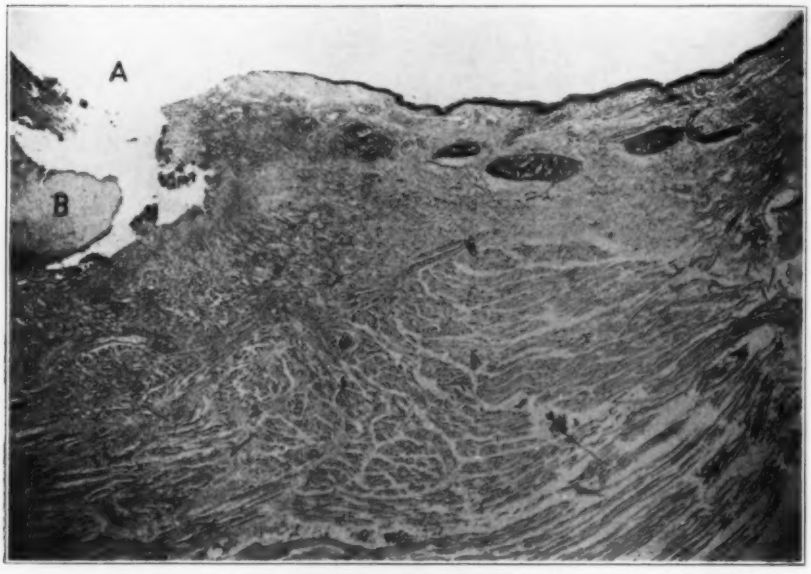

Fig. 3 (case 2).—Longitudinal section of a vocal cord, low power view. The anterior two thirds of the vocal cord and, posteriorly, the contact ulcer (*A*) are shown. The section was made in the subglottic region; it shows no other pathologic change except the mild degree of inflammatory infiltration in the region of the mucosal glands. A part of the cavity of the ulcer was occupied by the denuded vocal process of the arytenoid (*B*).

those in the preceding case. The hypopharynx, epiglottis, ventricular bands and subglottic region were apparently normal. No pathologic changes were observed in the anterior third of either cord. In the region of the vocal process on each side there was an ulcer with a thickened edge; the cavity was about 1 mm. deep, and the denuded vocal process projected from the bed; when it was touched with a probe, the process was found to be denuded.

Histologic examination of sections made lengthwise permitted examination of the whole cord, the ulcer and the arytenoid cartilage (figs. 3 and 4). Under low magnification (fig. 3) a mild degree of inflammatory infiltration was seen around

the glands in the subglottic mucosa. In the neighborhood of the arytenoid cartilage there was an ulcer in which was seen the vocal process of the arytenoid. The cartilage was almost completely calcified, and superficial necrotic erosion was noted. The walls of the cavity of the ulcer were made up of chronic inflammatory tissue with areas of necrosis. The zone of granulation tissue around the ulcer was about 1 mm. in thickness, and nearby there was a slight inflammatory infiltration extending into the muscular fibers of the cords. All the inflammatory tissue in the neighborhood of the ulcer was of nonspecific type and was entirely lacking in any of the characteristics of a tuberculous lesion. On summing up the histologic findings, there was in this case a localized chronic inflammatory process of the vocal cords located around the vocal processes of the arytenoids, with cartilaginous necrosis as well as ulceration of the soft tissues. The lesions were typically symmetrical.

Fig. 4 (case 2).—Higher magnification of section shown in figure 3. The histologic character of the contact ulcer of the larynx (*A*) is shown clearly. The walls of the ulcer were covered with chronic inflammatory infiltration with superficial necrosis. The vocal process of the arytenoid cartilage was denuded and projected into the cavity of the ulcer; it also was superficially necrotic (*B*).

COMMENT

The foregoing cases are interesting because they permit for the first time a pathologic demonstration of the correctness of the original clinical observations of Chevalier Jackson on contact ulcer of the larynx. In case 1 the patient died of pulmonary tuberculosis, but nevertheless the symmetrical ulcerative lesions of both vocal cords were distinctly nontuberculous. In fact, the localization as well as the type differed from

those in laryngeal tuberculosis. The histologic picture was distinctly that of chronic ulcerative nonspecific inflammation.

In tuberculous patients it is not uncommon to find chronic nonspecific inflammation of the larynx produced by the trauma of coughing and by the passage of secretion full of pyogenic organisms. One of the supposed routes of tuberculous infection of the larynx is the implantation of tubercle bacilli on a chronic inflammatory lesion.

In case 2 there was the same condition of a localized inflammatory lesion with ulceration in the neighborhood of the arytenoid cartilage and superficial necrosis of the protruding vocal process of the arytenoid cartilage. In this case also the lesion was perfectly symmetrical. In both cases the ulcerative process was strictly localized, with no other point of erosion or gross evidence of inflammation anywhere in the larynx. In both cases the ulcerative lesion was still advancing, showing active necrosis in the walls of the ulcer as well as in the vocal process of the arytenoid cartilage.

In neither of the two cases was there anything in the history, such as vocal abuse, that would bear on the etiology. It is, of course, impossible to demonstrate positively in these cases that the ulceration of the mucous membrane depended on protrusion or erosion of the cartilage as a cause; it seems improbable that the necrosis was a primary process. It seems probable, however, that the perpetuation of the disease and its chronicity were due to the necrotic condition of the cartilages at the tip of the vocal processes, producing trauma during every phonatory act.

MECHANISM INVOLVING FOREIGN BODIES

IN THE POSTCRICOID NARROWING, WITH SPECIAL REFERENCE TO SHARP BODIES SUCH AS OPEN SAFETY-PINS

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Jackson,[1] in his recent compilation of foreign bodies, has found that safety-pins are third in the frequency of all bodies he has removed from the respiratory and esophageal tracts: viz., bones in 350, coins in 306 and safety-pins in 128 cases. In the otolaryngologic clinic within a period of seventeen days we have had to remove, besides other foreign bodies, safety-pins from three patients in whom the pin obstructed the postcricoid narrowing.

Discussions in articles and popular textbooks bearing on this subject tend to emphasize the nature of the foreign body and its removal. Haslinger,[2] Waldapfel[3] and Wiethe[4] of Hajek's clinic and Abel[5] likewise laid great stress on every form of mechanism and on the manner of removal of foreign bodies from these regions, but neither they nor any other authors, I have found, make reference to the interesting theory and anatomicophysiologic proof regarding conditions which make it possible for safety-pins with their open ends facing downward to become lodged at such a low level as the postcricoid narrowing rather than to become embedded in any of the soft parts higher up.

There is no need to discuss the simple mechanism of the usual variety of blunt or fine pointed bodies which, if large, remain at certain natural narrowings, or, if small, follow the line of least resistance, as is shown by their preponderance in the right bronchus as compared to the left. Ordinary physical factors account for this majority, but in our particular questioning we have correlated interesting anatomicophysiologic data which clearly answer the requirements of this mechanism.

Presented April, 1932, at a conference of the Otolaryngological Department, Research and Educational Hospital, University of Illinois, College of Medicine.

1. Jackson, Chevalier: Foreign Bodies, The Cyclopedia of Medicine, Philadelphia, F. A. Davis Company, 1932, p. 486.

2. Haslinger, F.: Personal studies and communications in Vienna, 1931.

3. Waldapfel, Richard: Personal studies and communications in Vienna, 1931.

4. Wiethe: Personal studies and communications in Vienna, 1931.

5. Abel, A.: Oesophageal Obstruction: Its Pathology and Treatment, New York, Oxford University Press, 1929, p. 3.

Briefly, the hypopharynx and upper orifice of the esophagus are formed by the lower fibers of the inferior constrictor muscles of the pharynx. Posteriorly, this muscle, together with the middle and superior constrictors, inserts in a common tendinous raphe, the median pharyngeal raphe, and is separated by the loose pharyngobasilaris fascial sheath from the bodies of the cervical vertebrae in the midline and from the longus colli and capitus muscles laterally. It is its origin which concerns us most, not so much its lateral origins from the cricoid (m. cricopharyngeus) and thyroid (m. thyropharyngeus), but its anterior origin from the cricoid plate. Abel [5] emphasized this origin as being "inadequately, often inaccurately, described by the ordinary textbooks, although Birmingham, in 1898, has given the most satisfactory anatomic description of this region, which I have verified by several dissections," and which I have further confirmed clinically.

To quote Abel and Birmingham:

> The outer or longitudinal muscular fibers form over the oesophagus generally, with the exception of its upper end, a well-developed continuous layer of longitudinal bundles, running parallel to one another, and in the direction of the tube. This layer, which is considerably thicker in general than the circular layer, will be distinctly displayed if the connective tissue which lies outside the muscular coat be carefully and completely removed from the oesophagus, which has been moderately distended and still retains its connections with the larynx.
>
> On viewing the posterior aspect of such a preparation it will be seen that the longitudinal fibers on this surface form a continuous layer until a point one to one and a quarter inches below the cricoid is reached. Here the longitudinal fibers on the back of the tube divide into lateral halves, which gradually separate from each other and wind around to the side, leaving a V-shaped interval between the diverging masses above. As they pass around to the sides, these fibers form *two stout bands*, the bundles of which run obliquely or somewhat spirally upwards and forwards. They ascend beneath the lower margin of the inferior constrictor, and, having been joined by the longitudinal fibers of the side and front of the gullet (pharynx and oesophagus), they wind round to the front, where, meeting, they end below the middle of the back of the cricoid on a *stout flat tendon* through the medium of which they are attached to the cricoid cartilage. The tendon, which is about ¼ inch in width above and somewhat wider below, runs up over the back (lamina cricoidea) of the cricoid, and having been joined by certain other fibers (inferior constrictor pharyngis) it is finally attached to the upper end of the vertical ridge of the cricoid plate, near the point at which this ridge passes into the upper border of the quadrilateral plate. (Italics by author.)

During respiration, the orifice of the esophagus (the lower part of the hypopharynx) is in a normal state of contraction, so that the cricoid plate rests directly on the posterior wall of the hypopharynx, which assumes its characteristic slitlike appearance with its laterally placed culdesacs, the piriform sinuses.

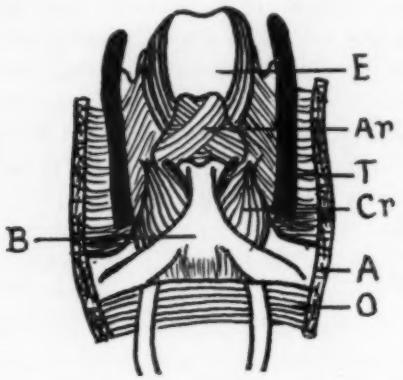

Fig. 1.—Posterior laryngeal and anterior esophageal musculature, showing the common tendinous raphe. *B* indicates a common tendinous raphe (median pharyngeal raphe); *E*, the epiglottis; *Ar*, the arytenoid muscles; *T*, the thyroid cartilage; *Cr*, the posterior crico-arytenoid muscle; *A*, the inferior pharyngeal constrictor muscle, and *O*, the circular muscle fiber of the esophagus.

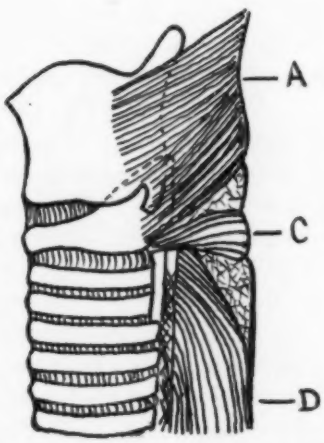

Fig. 2.—Lateral view of the inferior pharyngeal constrictor and the longitudinal fibers of the esophageal muscles. *D* indicates the longitudinal muscle fibers of the esophagus, approaching the posterior surface of the cricoid cartilage, to form a common tendinous raphe; *A*, the inferior pharyngeal constrictor muscle, and *C*, the horizontal fibers forming the cricopharyngeus muscle, or the superior sphincter of the esophagus.

What happens during the act of swallowing? Abel [5] discussed very well the esophageal reflexes; Cunningham [6] and others divide the act into an involuntary and voluntary stage, but we agree with Howell [7] that Magendie's classic description, dividing it into three stages, is most correct. Corresponding to the three anatomic regions, the mouth, pharynx and esophagus, the first represents an excellent example of a finely coordinated movement, mostly voluntary, while the two latter are entirely involuntary reflex in their character.

It is our purpose to correlate the foregoing anatomic structures with the following physiologic data to explain an interesting problem, and it is best to stress at this point that we are concerned only with the first stage entirely and the beginning of the second, before any bolus enters into the esophageal lumen or before the pharynx contracts.

The first stage, voluntary, consists in the passage of the bolus of food through the isthmus of the faucies, which is ascribed to the action of the cheek muscles (buccinator) and the muscles of the tongue itself. Howell and Cunningham agreed in their description of these stages. The pharynx being a common passage for the air and food, it is important that this part of the act be consummated quickly. According to the older description, the motor power driving the bolus downward through the pharynx is derived from the contraction of the pharyngeal muscles, particularly the constrictors, which contract from above downward and send the food into the esophagus. Kronecker and Meltzer,[8] and Cannon and Moser [9] later, however, have shown that the contraction of the mylohyoid muscle in the floor of the mouth is the most important factor in this act of shooting the food suddenly through the pharynx into the esophageal orifice (the mylohyoid also raises the larynx through their common connection with the hyoid bone). The contraction of this muscle marks the beginning of the purely involuntary part. The bolus of food lies on the dorsum of the tongue, and by the pressure of the front of the tongue against the hard palate, it is shut off from the front part of the oral cavity. When the mylohyoids contract sharply, the bolus is put under pressure and is shot into and through the pharynx. This effect is aided by the contraction of the hyoglossal muscles, which, by moving the tongue backward and downward, tend to increase the pressure put on the food. Simultaneously, the muscles of the soft palate (levators and tensors veli palatini, uvulae, glossopalatini and pharyngopalatini) tense and approximate it against the posterior wall of the pharynx, thereby closing

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6. Cunningham: Text-Book of Anatomy, ed. 5, New York, William Wood & Company, 1930, p. 467.

7. Howell, W. H.: Text-Book of Physiology, ed. 8, Philadelphia, W. B. Saunders Company, 1923, p. 709.

8. Kronecker and Meltzer, quoted by Howell.[7]

9. Cannon and Moser, quoted by Howell.[7]

off the nasopharynx; and the adductor and sphincter muscles of the larynx contract to close the glottis. The larynx is raised by the mylohyoid and levator pharyngeal muscles against the epiglottis. The anterior attachments of the hypopharynx and upper orifice of the esophagus to the cricoid are pulled up with the larynx away from the posterior wall, widening the piriform sinuses anteroposteriorly, so that instead of being slitlike, the hypopharynx becomes quite a large receptacle for the bolus.

Before the second stage begins,[7] in the case of liquids and small bodies, the bolus is shot through the whole length from the oral and mesopharyngeal cavity, even through the whole length of the esophagus, by the force of the initial act of swallowing.

SUMMARY

We therefore conclude: (1) that objects of small mass, comparable to liquids, such as safety-pins, are not grasped by the pharyngeal constrictors, but are quickly and forcibly propelled through the mouth and mesopharynx; (2) that the hypopharynx, because of its attachments anteriorly, is open and ready to receive objects, rather than closed, permitting rapid descent into the postcricoid space. These two factors together account for the failure of safety-pins the open ends of which are pointed downward to become embedded at a more plausible higher level.

The following three cases are briefly presented as typifying the usual histories and findings encountered in foreign bodies of this type.

REPORT OF CASES

CASE 1.—Miss B. Z., aged 13, was brought from La Salle, Ill., on April 22, 1932, with a history of pain in the throat and difficulty in swallowing after aspiration of a safety-pin. The roentgenogram (fig. 3) revealed an open safety-pin at the level of the sixth cervical vertebra, the open ends pointing downward. The lateral view showed the safety-pin, points open downward, lying with its broad surface in the lateral plane, revealing at once its location in the hypopharynx or esophagus rather than in the larynx. Indirect laryngoscopy revealed the pointed branch lying in the postarytenoid space and the keeper branch hidden in the postcricoid space. The esophagoscope was inserted into the postcricoid space, and the pin was removed by pulling it through the tube.

CASE 2.—Miss J. M., aged 36, was admitted on April 30, 1932, with a history of pain in the throat and inability to swallow, following aspiration of a safety-pin. A roentgenogram (fig. 4) revealed an open safety-pin with its points open upward, lying at the level of the sixth cervical vertebra, and the anteroposterior view showed it to be lying in the postcricoid space, rather than in the laryngeal lumen. The pin was removed after some difficulty through the esophagoscope. The patient made an uneventful recovery. (Courtesy of Dr. F. L. Lederer.)

CASE 3.—Baby C. H., aged 6½ months, was admitted on May 9, 1932, with a history of an attack of choking and coughing and disappearance of a safety-pin which had been used to pin some medals on the baby's clothes. A roentgenogram

Fig. 3.—Roentgenogram in case 1.

Fig. 4.—Roentgenogram in case 2.

(fig. 5) showed an open safety-pin, ends facing upward, at the level of the seventh cervical vertebra. Extraction of the pin was most difficult, the pointed end being found embedded in the esophageal mucosa in the postcricoid space. Removal was obtained by version in plane of expansion, according to Jackson.[10] (Courtesy of Dr. F. L. Lederer.)

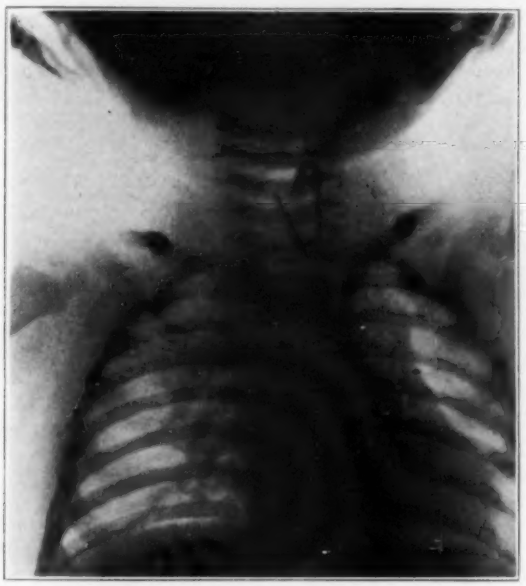

Fig. 5.—Roentgenogram in case 3.

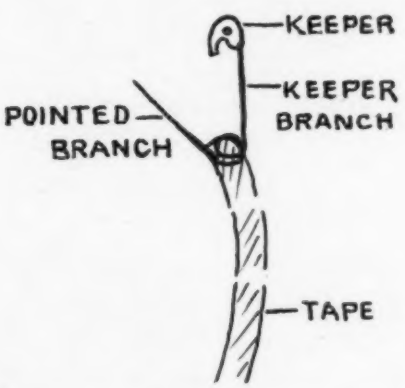

Fig. 6.—Simple but expedient addition to a safety-pin as a prophylactic measure.

DISCUSSION

Dr. Francis L. Lederer: This is an intriguing physiologic and anatomic revelation. Clinically, it presents many unpleasant factors to the laryngologist who

10. Jackson, Chevalier: Bronchoscopy and Oesophagoscopy, ed. 2, Philadelphia, W. B. Saunders Company, 1927, p. 250.

is faced with the problem of removal, particularly when pointed objects are found embedded against the pathway of removal. I feel that there is an added prophylactic problem, which could be partly, if not greatly, solved by the simple addition of a band of tape sewed to the spring of the most common offender, the safety-pin, and I have recommended its use whenever I find myself in the position to do so. Accidental aspiration of a pin prepared in this manner would not be so serious a complication to the victim. Its removal would certainly be greatly facilitated, as one would have only to grasp the loose end of the tape to accomplish its removal. Another prophylactic measure might be considered in making safety-pins with springs normally bringing the two branches to the closed position rather than the open.

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DERMOID CYST OF THE NASAL DORSUM

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Dermoid cysts of the nasal dorsum are rare. There are a few cases reported in the foreign literature, but only one in the American.

The rarity of the condition has induced me to report the following case.

REPORT OF CASE

History.—Mrs. E. L., white, a housewife, aged 30, complained of a continuous discharge of bloody pus and hairs from the region of the dorsum of the nose. She made no other complaint. She averred that she had always noticed that her nose was broadened and elevated at its upper portion, but that she never had any physical discomfort from it. About six years before the present examination, the elevation increased considerably; this worried her, and she consulted the family physician, who told her that it was a cyst which had to be removed. A surgeon opened it, a local anesthesia being used, and a fistula resulted from the operation. The patient did not consult any physician during the ensuing six years, because she did not have any trouble, except that it was necessary to remove the pus and pull out the hairs.

Examination.—The patient was a robust woman. The nose appeared to be normal except for the slight deformity at its bridge. The region at the junction of the middle third with the upper third of the nasal dorsum was broadened, elevated and striped by a longitudinal linear scar about 2 cm. long. No opening was perceptible. I asked the patient to show me where the discharge was coming from, and, as she ran her finger down along the nasal dorsum, a considerable amount of yellowish bloody discharge came out of a pinpoint opening located at the lower border of the nasal bones. After the pus was wiped away, a black hair was noticed coming out of the opening. With a pair of forceps I pulled out the hair, which was immediately followed by another one. At that first visit I pulled out about one dozen hairs. The opening was circular, very small and without any redness or swelling around it.

Palpation revealed a soft area of about 2 sq. cm. which was fluctuating. This area extended from the lower border of the nasal bones upward, and the bones seemed to be separated for a length of about 2 cm. and a width of 1 cm. At the slightest touch applied on this soft area, bloody discharge and hairs would come out of the opening. No tenderness could be elicited in the area and in the vicinity of it. A probe introduced through the opening could be pushed upward for about 2 cm., downward for about 1 cm., and laterally for about 0.5 cm.

The intranasal examination did not show anything abnormal, except a thickening of the nasal septum about the region of the cyst.

The pus squeezed out of the fistula developed a pure culture of Friedländer's bacillus (secondary infection).

The roentgen studies did not add much information.

Read before the Philadelphia College of Physicians.

Fig. 1.—In *A*, the location of the fistula is indicated by the arrow; in *B*, by the black dot. Above it the linear scar is seen.

Fig. 2.—Photographs of casts taken before and after operation. The upper one was taken during the operation, and it shows the exact size of the fistula. The lower one was taken before: hairs can be seen emerging from the fistula.

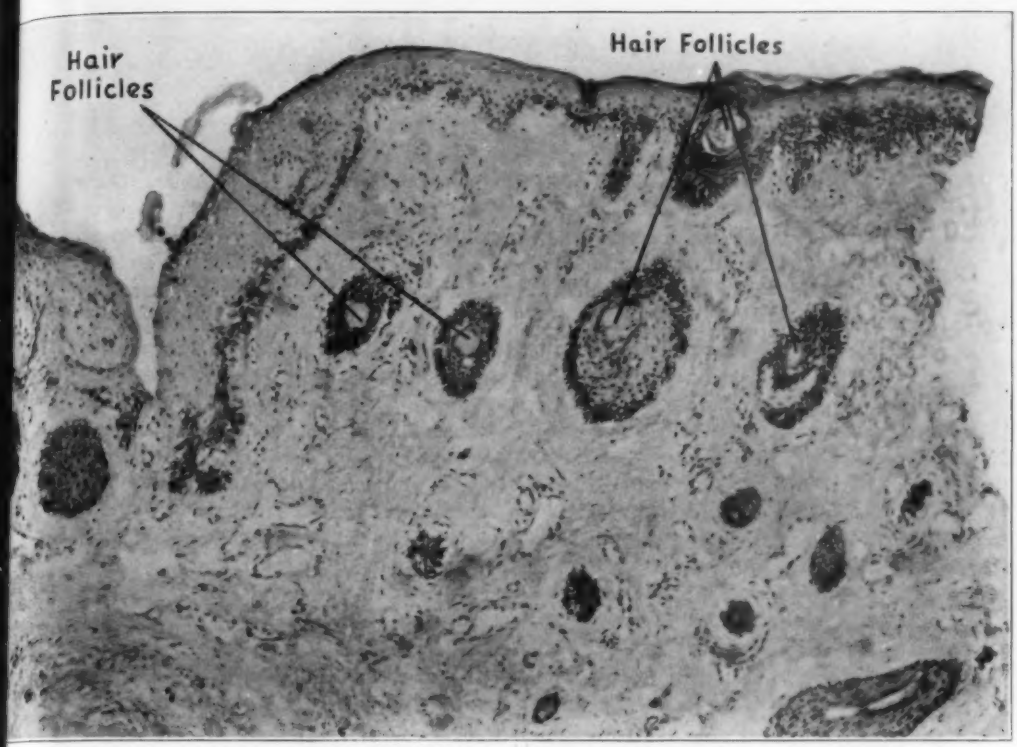

Fig. 3.—Photomicrograph of the specimen. I was unable to find a field where sebaceous glands could be demonstrated at the same time.

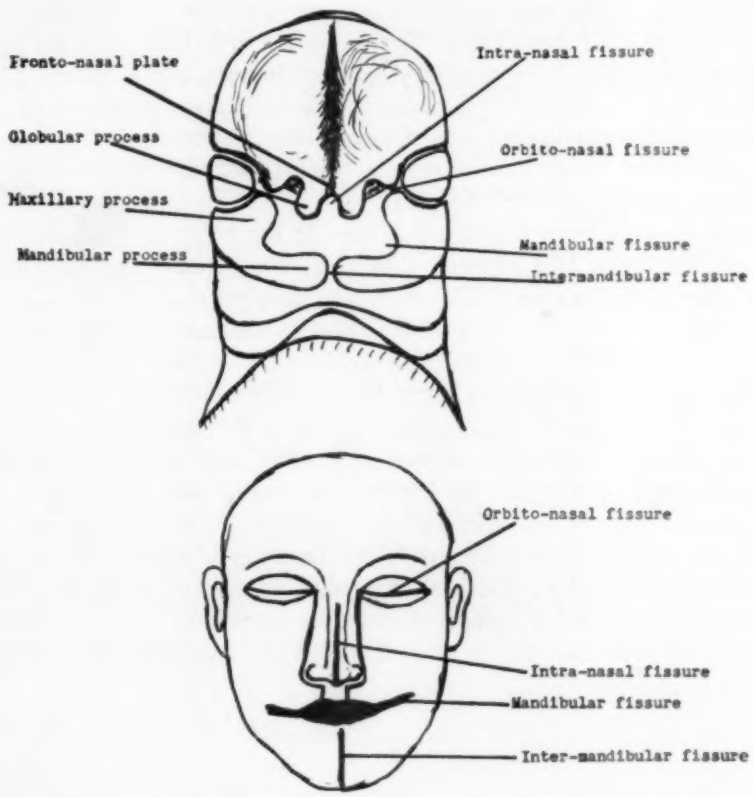

Fig. 4.—The fissures and their locations. (After Bland-Sutton.)

I tried to introduce iodized oil through the opening, but I failed twice. Only about 0.5 cc. could be forced into the cavity even after the content of the cyst was milked out. The roentgenograms were indefinite in establishing the extension of the tumor; they suggested extension into the frontal sinuses, the ethmoids and the area of the cribriform plate. The displacement method of Proetz did not give any definite information.

Operation.—I decided to explore the cavity. Using local anesthesia, I incised the skin and immediately encountered an oval cavity the size of a cherry, which was full of cheesy yellowish material and loose hairs. Immediately under the skin the edges of a sac were easily differentiated. The inside of the sac and the fistular tract were covered with growing hair. The bony wall was intact. I closed the wound tightly and injected iodized oil into the cavity for further roentgen studies, which reassured me that the cavity was limited to the area explored and that it did not have any communications beyond. A few days later I carefully dissected out the sac, which was lying in a bony nest, was oval and the size of an olive. I closed the wound, leaving a small cigaret drain in its lower part, and the patient made an excellent recovery with good cosmetic results.

Laboratory Report.—The specimen consisted of four fragments of white fibrous tissue with hairs adherent and a few bluish bands running through them.

Microscopically, there was found squamous epithelium overlying fibrous and fatty tissue which contained a few sebaceous glands and many hair follicles.

Dermoid cysts occur in and around the nasal area in certain definite positions, such as the bridge of the nose, along the nasal dorsum (most commonly between the lower margin of the nasal bones and the tip), near the tip of the nose and at the inner angle of the orbit and the nasofacial sulcus. In order to appreciate the origin of dermoid cysts in these situations, it is necessary to bear in mind the relation of the facial fissures in the embryo, which in the adult are represented by the orbits (orbitonasal fissure), the lacrimal ducts (persistent portions of the orbitonasal fissures), the mouth (mandibular fissure) and certain furrows in the lips and cheeks (fig. 4).

The dermoid cysts which are located in the areas of the embryonal fissures are formed by sequestration of portions of the surface epithelium during the process of closure of the fissures.

In the area in which the cyst is located in my case there are not any fissures. It is believed that the dermoid cysts in this region (and in the region of the bridge) are formed in the following way: In the early embryo the frontonasal plate, which forms the nose, consists of a lamina of hyaline cartilage covered externally with skin and internally with mucosa. After the third month of embryonal life, bony tissue extends in between the skin and the cartilage. This bony tissue will form the nasal bones. The cartilage becomes absorbed during this process of ossification. During the gradual separation of the skin from the cartilage of the frontonasal plate by the intrusion of the nasal bones, small portions of the skin or epithelium become sequestrated and develop into dermoid cysts.

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EXPERIMENTAL SURGERY OF THE NOSE AND SINUSES

III. RESULTS FOLLOWING PARTIAL AND COMPLETE REMOVAL OF THE LINING MUCOUS MEMBRANE FROM THE FRONTAL SINUS OF THE DOG

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It is common when operating on the sinus to remove the lining mucous membrane completely. For that reason it is of value to learn how the sinus heals after such a procedure. Considerable interest has been aroused in this subject recently, and discussions show some difference of opinion. It has been commonly believed that sinuses so treated filled with scar more or less completely. Mosher and his co-workers [1] and Knowlton and McGregor [2] have shown that the mucous membrane regenerates very well in some cases. Semenov and Kistner [3] found that regeneration of epithelium was variable. They found that formation of scar was marked after complete removal and sometimes this scar was devoid of epithelial covering. Coates and Ersner [4] reported an experiment on a dog in which there was regeneration of epithelium after complete removal. In all probability, when the whole story is known, these apparently paradoxical results will be found to be consistent.

This paper concerns the results obtained in two series of experiments undertaken in connection with those reported in a previous article.[5]

Part of a paper read before the American Laryngological, Rhinological and Otological Society, Atlantic City, N. J., May 23, 1932.

Work done in the Division of Experimental Surgery and Pathology, the Mayo Foundation, Rochester, Minn.

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In the first series the mucous membrane of ten frontal sinuses of eight dogs was removed entirely. In the second series, large portions of the mucous membrane of four sinuses of four dogs were removed, but some islands were left intact. The process and results of healing were observed for many months. Some of the dogs are still living and therefore postmortem observations are not available in all.

TECHNIC OF OPERATION

The operations were confined to the frontal sinus. All dogs were under anesthesia with ether when operated on, and an aseptic, surgical technic was employed. Both sinuses were regularly exposed through a median line incision over the frontal bone, and enough of the roof of each sinus was removed to allow the necessary surgical manipulations within.

The normal lining mucous membrane is loosely attached to the bone in most regions, but in a few places it is firmly attached. In removing it for the purposes of these experiments, it was peeled out as cleanly as possible, using a curet and sponge to free the adherent spots. Ordinarily the ostium was not disturbed. When it was intended to leave islands of epithelium intact, these were outlined by a scalpel before the rest of the membrane was removed. At the end of the operations, the wound was closed by stitching the incision in the scalp.

At subsequent operations, for purposes of observation, if the sinuses were filled with scar tissue, this was explored by incising it deeply with a scalpel. As some small cysts may have been missed in this way, it is possible that the two sinuses reported as completely obliterated may have contained cysts and should have been grouped otherwise.

The time during which observations were made varied from a few weeks to more than a year. For the purposes of these experiments, each dog was observed by surgical exploration from two to eight times.

RESULTS

In general, the process of healing resulted in the formation of massive scars that tended to obliterate the sinuses. In the first series of ten sinuses which were completely denuded of mucous membrane, results were somewhat variable (see table). Heavy scar tissue formed in all sinuses. In two dogs, the sinuses seemed to be entirely filled with scar, leaving no vestige of a cavity (dogs 1 and 2, left sinus). In dog 2 (left sinus) the scar was partially ossified. Four sinuses were filled with scar tissue and completely obliterated except for an air cavity at the ostium. In one of these sinuses (dog 8) the cavity was about a quarter to a third the size of the original sinus (fig. 1). In the other three sinuses it was tiny. In one of the three (dog 7, right sinus) it seemed to vary in size. Three weeks after denudation it occupied about a fourth of the original sinus. At the end of six months it seemed to be much larger, occupying from a third to a half, but after ten months it was very small again. The scar in the posterior portion of this sinus became ossified. The other four sinuses were

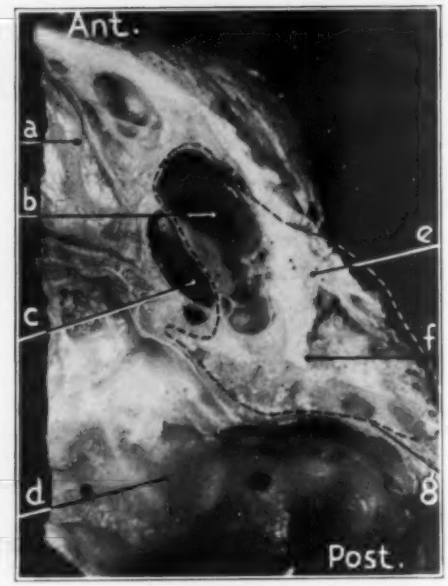

Fig. 1.—Sagittal section through the frontal sinus of dog 8, about a year after removal of the lining mucous membrane: *a*, nasal cavity; *b*, reformed sinus; *c*, ethmoid cell; *d*, cranial cavity; *e*, massive scar; *f*, scar partially ossified; *g*, outline of original frontal sinus. It was thought, at the time of operation, that the membrane was completely removed. If it was, this cavity, which is about a third the size of the original sinus, represents true restoration.

Fig. 2.—The right frontal sinus of dog 5, eleven weeks after complete denudation, as seen from the lateral aspect after a wedge of bone and scar was sawed out. The original sinus is outlined by the dotted line. A cyst filled with mucin may be noted in the anterior portion. Most of the scar posterior to the cyst was ossified.

filled with scar tissue and obliterated except for cysts filled with mucin. Two cysts were found in one sinus and one in each of the other three sinuses. In one animal (dog 5) the scar was partially ossified (fig. 2). The procedures and results are shown in detail in the table.

In the second series of four sinuses of four animals, some islands of mucous membrane were purposely left at the time of denudation. In healing, these sinuses also gave evidence of massive formation of scar. In two sinuses (dogs 9 and 10) cysts developed that contained thick mucin where the epithelium had been left. The former of these

Results of Ten Experiments in Which the Frontal Sinus Was Completely Denuded of Mucous Membrane

Dog	Side Operated On	Times Operated on Subsequently for Observation Only	Under Observation, Weeks	Results
1	Left	1	12	Sinus completely obliterated by scar tissue
2	Left	2	53	Completely obliterated by scar tissue, which was partially ossified
2	Right	2	53	Filled with scar, which contained one cyst filled with mucin
3	Left	7	45	Filled with scar, which contained one cyst filled with mucin
4	Left	5	46	Filled with scar, which contained two small cysts filled with mucin
5	Right*	1	9	Filled with scar, which contained one fairly large cyst filled with mucin
6	Right	3	42	Obliterated by partially ossified scar, except an air cavity next to the ostium
7	Right	5	43	Obliterated by partially ossified scar, except a small air chamber at the ostium
7	Left†	1	4	Posterior two-thirds obliterated by scar; there was an air chamber at the ostium occupying almost a third of the sinus
8	Right	4	42	Posterior two-thirds filled with partially ossified scar; air chamber at ostium occupied the anterior third

* This sinus had become infected following an operation at which the ostium was closed. It drained for a while through a fistula and was then operated on and completely denuded of mucous membrane.

† This sinus had been unroofed and had been exposed to the outside air for more than a year; it was denuded and the scalp closed over it. When opened subsequently, there were signs indicating that some of the mucous membrane near the ostium had been missed.

animals was under observation thirty-eight weeks, and the latter forty-one weeks. In three sinuses (dogs 10, 11 and 12) partial restitution of the sinus cavity was shown. Results were not always uniform, nor exactly as expected. In one animal (dog 10) the posterior three fourths of the sinus was denuded. Instead of filling with a solid mass of scar as most of the others did, this one healed with thin bands and diaphragms in the major portion, and eventually became fairly well restored. After nine months there remained a rather heavy scar in the posterior third, which contained a cyst filled with mucin. The rest of the cavity was open to the ostium, and apparently was well lined by epithelium. The posterior two thirds of the sinus of one animal (dog 11) was denuded and the animal observed for forty-five weeks. This portion became covered with a thick, fibrinous exudate

that seemed to build up from the walls in a manner similar to the formation of the diaphragms resulting from removal of circular strips [5] (fig. 3). This fibrinous exudate eventually became organized into dense connective tissue. As it contracted, it formed a cavity, the fundus of which was hemispheric; the cavity was wide open anteriorly into the undisturbed anterior third of the sinus. This cavity was much smaller than the portion of the original sinus that it occupied. It did not conform to the contour of the wall of the sinus. It did not become lined by epithelium even after almost a year, although it was easily accessible to the epithelium in the anterior third of the sinus (fig. 4). A spot of a finely divided suspension carbon, placed in it for testing ciliary action, remained unmoved in the same position for ten months, and at microscopic examination proved to be incorporated with the connective tissue like a tattoo mark. During an entire year, as seen at six subsequent observations, this cavity remained empty, clean and intensely white. It was apparently almost avascular. In other words, the posterior, denuded two thirds of the walls of this particular sinus, in healing, became thickly covered with a mass of heavy scar tissue that contracted in such a way as to form the cavity described. The only change that occurred from month to month was that this mass of scar tissue slowly receded in the direction of the fundus of the cavity; as a result, the cavity became somewhat smaller.

The sinus of one animal (dog 12) was denuded throughout, except that several islands of epithelium were left in place. The period of observation was four weeks. In healing, this sinus acted like the previous one. Heavy, thick, white connective tissue that contracted to form a more or less spherical cavity covered all the denuded areas. The epithelial islands remained as deep pockets filled with mucin in the walls of this cavity (fig. 5). Ciliary motion and power remained excellent within these pockets, but the mucin could not escape because the surrounding connective tissue was devoid of epithelium and therefore afforded no ciliary action. The epithelial islands in this case did not reline the sinus, although it remained an open cavity. As in the previous experiment (dog 11) the fibrous walls were clean, white and smooth. The sinus did not become inflamed.

Under certain conditions, this scar tissue undoubtedly does become covered with epithelium. In order to determine this with certainty, the restored roofs of six sinuses were carefully studied with this point in mind. A large portion of the roofs of these sinuses had been removed completely, with the exception of the scalp. In healing, the defects in the bone became closed by a diaphragm of heavy scar tissue that became partially ossified in most cases. The epithelium regenerated to cover the under surface of these scars. Unquestionably this was the

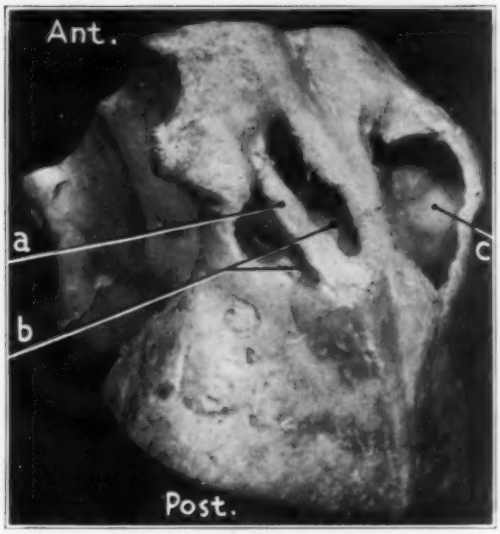

Fig. 3.—The appearance of an incomplete diaphragm of scar tissue that had formed in the left frontal sinus of a dog fifteen weeks after the removal of a circular strip of mucous membrane, 7 mm. wide: *a*, diaphragm with perforation; *b*, divided cavity of the left frontal sinus; *c*, normal right frontal sinus. The roofs of both frontal sinuses have been largely removed, and the lateral wall of the left sinus has been cut down in order to show the opening through the incompletely formed diaphragm. The portion of sinus lateral to the diaphragm was unable to drain properly, and contained an accumulation of mucin. A series of such experiments is reported in a previous article.

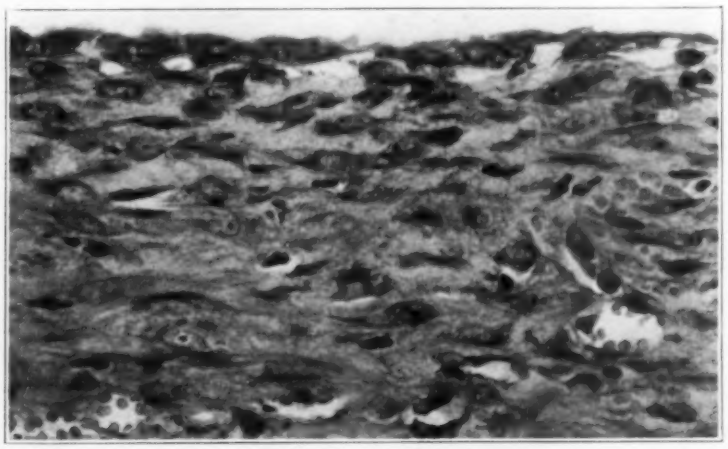

Fig. 4.—The structure of lining of the denuded portion of the left frontal sinus of dog 11, sixty-four days after removal of the mucous membrane. It is composed of smooth, uninflamed connective tissue, devoid of epithelium. The same conditions obtained in this cavity at the end of ten months, even though the surface of this scar tissue was readily accessible to the epithelium lining the undisturbed anterior portion of the sinus.

result of true regeneration and not merely of incomplete removal, because the entire roof was removed with all its layers except the scalp. In all cases the epithelium appeared to be normal. Sometimes it lay directly on dense connective tissue, almost devoid of blood vessels, and sometimes it lay on a rather loose submucosa, containing a good supply of vessels. Although the defect in the roof may have been 25 or 30 mm. across, regeneration of the epithelium was complete in some cases after two weeks.

In one animal (dog 9), the mucous membrane was stripped out completely, with the exception of an island in the deepest part of the sinus. In healing, the sinus became filled with a mass of scar tissue

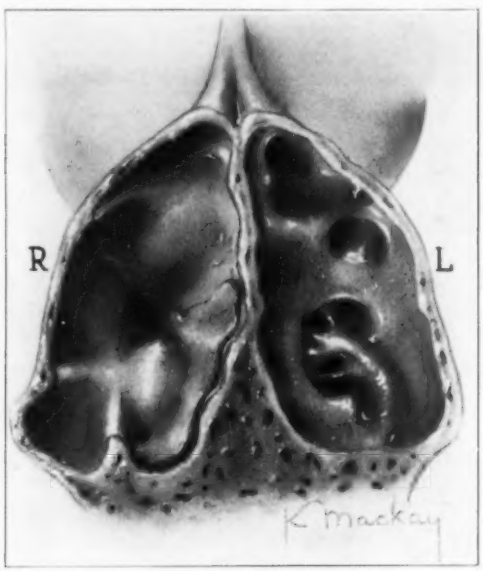

Fig. 5.—The interiors of the frontal sinuses of dog 12, as found at necropsy after complete removal of the roofs by sawing in a plane parallel with the frontal bone (anterior view). The left sinus had been denuded of mucous membrane four weeks previously, except for three islands represented by the three roughly circular pits. The pits were lined by good epithelium, and the plateau between was formed by white scar tissue devoid of epithelium.

containing several cysts filled with mucin. It looked much like the sinuses of the first series, which supposedly had been completely denuded, but which in healing filled with cysts that contained scar. In this case the cysts were larger.

The cysts are sufficiently interesting to deserve a little more detailed description. They varied considerably in size, the largest containing perhaps 0.5 cc. of mucin. Some were found attached to the bony wall, as would be expected when islands of epithelium were left intact.

Many, however, were found in the middle of the scar tissue at some distance from the walls. The mucin contained within them was brown, translucent and very thick. A mass of such mucin, laid on a dry surface, could be picked up with a pair of forceps, leaving the spot where it had lain practically dry. Cultures were made of the mucin contained in a number of the cysts. In all but a few it was found to be sterile. In those few from which positive cultures were obtained, contamination was strongly suspected. It is difficult to remove the mucin without contaminating it. The epithelium lining the cysts was found on microscopic examination to be of a moderately tall columnar, ciliated type, lying on a dense base of connective tissue. Apparently it was not under any pressure as might be expected in a cyst lined by epithelium containing active gland cells. Ciliary activity was very great. Ink placed on the surface moved at a high rate of speed. The greatest rate of ciliary flow found anywhere in any of my experiments was found within one of these cysts; it reached a speed of 24 mm. a minute.

COMMENT

If the main results of these experiments were to be given in a single statement, it would be: After complete denudation of the frontal sinus of the dog, the sinus tends strongly to heal by a massive formation of scar tissue that obliterates the cavity, but, as has been seen, the cavity was passably well restored in three instances. Why the epithelium covered the surfaces again so well in one of these instances and failed to do so in two others is not entirely clear. It is probably to be accounted for on the basis of nutrition; it is possible that some periosteum with a more or less intact blood supply was left. The heavy scar tissue that formed on the walls of the sinuses in two instances seemed to result from deposits of fibrin organized by fibroblasts, and had a poor blood supply. It is curious that such connective tissue can remain uncovered by epithelium, exposed to the air, and yet show no signs of inflammation. I am not aware that this happens with any other tissue.

The small cavities found at the ostium in some cases were probably the result of retraction of the mass of scar from the only point at which it was not attached, combined with the growth of epithelium from the ostium. Why some cavities became so much larger than others is a matter of speculation. Perhaps it was a function of nutrition, or perhaps the epithelium was not so completely removed in some cases as in others.

The cysts that were found embedded in the scar tissue were obviously all formed from epithelium that was left intentionally or otherwise. Their presence in so many sinuses that were supposed to be completely

denuded emphasizes the difficulty of being certain that all the epithelium is removed. The fact that so many of the cysts found were not in contact with the bony walls of the sinus suggests that loose fragments of epithelium lost in the blood clots were sometimes able to survive and grow.

It cannot be assumed from these experiments that the sinuses of man treated surgically act exactly the same after denudation of the mucous membranes. It is common knowledge, for instance, that they do not become obliterated so readily. I do not believe that the difference in species would make any essential difference in result, but such factors as the presence of infection, supply of blood altered through disease, openings for the purpose of drainage, retained blood clots and anatomic characteristics might all serve to alter the outcome. These are matters for future study. It is conceivable that the maxillary sinus of the dog may respond differently than the frontal sinus.[6]

CONCLUSIONS

1. When the normal frontal sinus of the dog is denuded of mucous membrane, and the scalp is sutured over it without drainage, the sinus usually fills with scar tissue that obliterates the cavity.

2. In exceptional cases there is partial restitution of the sinus, with regeneration of the lining epithelium.

3. Under some circumstances a smaller cavity forms, the walls of which are composed of thick, white connective tissue, devoid of epithelial covering, and over which epithelium apparently cannot grow. This connective tissue shows no sign of inflammation even if exposed to air.

4. Under other conditions epithelium will grow over this heavy scar tissue. In some instances it appears to lie directly on the scar tissue, and in other instances it lies on vascular submucosal tissue.

5. If portions of epithelium are left within the sinus, cysts filled with mucin form within the obliterating scar.

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6. Since this paper was written, Knowlton has read a paper before the American Academy of Ophthalmology and Otolaryngology in which he stated that the maxillary sinus of the dogs did not become obliterated after denudation.

IMMEDIATE TRANSPLANTATION ON DEFECTS DUE TO ACCIDENT

REPORT OF TWO CASES

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While early transplantation on traumatic defects bears some risk of infection, it has, on the other hand, the advantage of affording the tissues good nourishment before the scar formation sets in. I present herewith two cases in which immediate transplantation proved to be of value.

REPORT OF CASES

CASE 1.—*Defect on the Nose from a Dog's Bite; Repair by Free Transplantation.*

History.—A girl, aged 5 years, was bitten by a dog. First aid was given by the family physician.

The patient was examined by me the day after the accident. Aside from several superficial scratches on the face, a piece was missing from the nose at the right side of the tip (fig. 1). The defect included the entire thickness of the skin, the lower edge of the tip (alar) cartilage and the rim of the nostril.

The question arose as to whether the wound should be left to heal first and the plastic repair be done later, or whether transplantation should be done immediately. If the wound were left to heal first, this would have the advantage of creating a clean surface. However, it would have the following disadvantages: First, the nose would be disfigured for some length of time (considering the age of the patient), probably for years. Second, tension of the scar would influence the growth of the tip cartilage and corresponding parts of the nose. Third, the secondary shrinkage of the connective tissue developed during granulation of the defect would affect a margin of the surrounding healthy tissues, which by later transplantation would have to be excised, thereby enlarging the defect.

Transplantation on this unclean surface would offer some risk of infection, but, on the other hand, it would eliminate the aforementioned disadvantages. If the transplant was not successful, the defect would not appear any worse than before the operation. For the aforementioned reasons, I decided on immediate transplantation. Forty-six hours after the accident, I operated on the patient under general anesthesia.

Operation.—After surgical preparation of the field, the fine granulations were removed and the ragged edges of the wound were trimmed, a smooth surface with an even margin being provided. The skin of the vestibuli nasi around the defect of the tip cartilage was undermined, and with 00 chromic catgut it was brought together in order to provide lining and receiving surface for the lower part of the transplant. An exact pattern was made from the defect with sterile

transparent paper. A piece of skin of full thickness, the size of the pattern, was taken from the inner side of the right thigh and sutured into the defect with interrupted fine dermal sutures. Dry dressing was applied with a thin layer of rubber sponge over the gauze, and adhesive tape, in order to provide even pressure

Fig. 1.—A piece of the nose was bitten off by a dog.

Fig. 2.—Front and side views five months after transplantation.

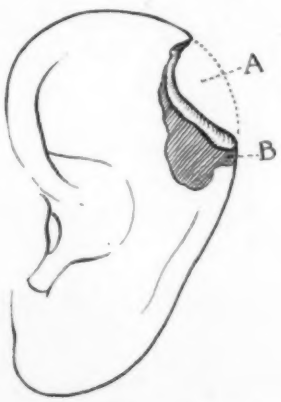
on the skin graft. Dressing after six days revealed good healing of the entire skin graft. The sutures were removed the same day.

The nose assumed its natural shape, and a few months after the operation evidence of repair was barely noticeable. Figure 2 shows the patient five months after transplantation.

CASE 2.—*Defect on Ear from Bite; Pedunculated Skin Transplantation from the Ear Itself, and Reconstruction of the Ear.*

History.—A man, aged 37, was bitten on the left ear in a row. He consulted me the following day. An irregular, elongated piece was missing from the upper part of the left ear (figs. 3 and 4). The lower half of the defect included only the anterior skin and part of the cartilage.

Fig. 3.—Part of ear missing owing to a bite.



Fg. 4.—Diagram of defect: *A*, missing area; *B*, denuded area.

As to the time for an operation, the same factors of scar formation and shrinkage were taken into consideration as in the previous case. However, as the risk of infection is greater on the ear than on the nose, I decided to wait a few days until the wound cleaned up and was covered with healthy granulations.

Operation.—Nine days after infliction of the injury, operation was performed, local anesthesia of 2 per cent solution of procaine hydrochloride and epinephrine being used. After surgical preparation of the field, procaine hydrochloride was

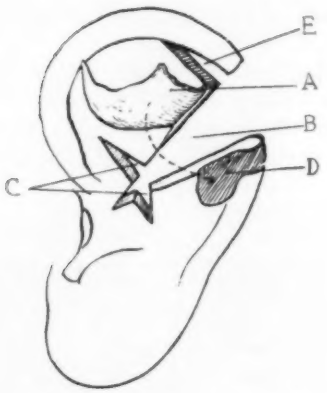


Fig. 5.—Diagram of operative procedure: *A*, elevated skin flap to cover denuded area (*D*); *B*, sector, including defect, excised through cartilage and posterior skin to decrease size of ear; *C*, wedge-shaped pieces excised near apex of sector; *D*, denuded area of lower part of defect, and *E*, wedge-shaped piece excised for alinement of reconstructed helix.

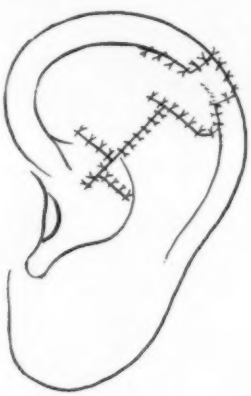


Fig. 6.—Diagram of reconstructed ear showing flap in place, covering lower part of defect, and suture lines.

Fig. 7.—Photograph of ear after operation.

infiltrated around the base of the ear. The granulations were removed, and the ragged edges of the skin were cut even. The anterior skin of the ear was elevated together with the perichondrium within a sector the base of which was the upper half of the defect. This elevated skin provided a flap (fig. 5*A*) to cover the denuded area of the defect (fig. 5*D*). The remainder of the sector, containing the cartilage and posterior skin, was excised (fig. 5 *B*). Two small wedge-shaped pieces were excised perpendicularly, near the apex of the sector, from the entire thickness of the ear to prevent it from curling forward (fig. 5*C*). From the elevated skin a flap was formed and turned downward to cover the denuded lower half of the defect. In order to bring the upper part of the helix in line with the lower, a wedge was excised parallel to the helix (fig. 5*E*). The edges of the wound were sutured with dermal sutures, including the skin and the perichondrium (fig. 6). Dressing five days after operation revealed healing by first intention. The sutures were removed consecutively up to ten days.

Examination three months after operation revealed a normal ear, and evidence of repair was scarcely noticeable (fig. 7).

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EFFECT OF EXTRACT OF THE SUPRARENAL CORTEX ON MAXILLARY SINUSITIS IN THE RABBIT

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Many studies on the pathology of the suprarenal glands have lent support to the theory advanced forty years ago that the suprarenal cortex detoxifies, probably by its lipids, not only metabolic products but also bacterial toxins, and also protects the organism against bacterial invasion. In 1889 Roux and Yersin,[1] the discoverers of diphtheria toxin, observed marked hyperemia of the suprarenal cortex of the rabbit and the guinea-pig following the subcutaneous or intraperitoneal injection of diphtheria toxin. The following year Behring,[2] in immunizing guinea-pigs against diphtheria toxin, found that the suprarenal cortex was always swollen and congested. Oppenheim and Loeper,[3] in their experimental and clinical investigations on diphtheria, tetanus, anthrax and pneumonia, found necrosis of the cortical cells, hemorrhage and polymorphonuclear infiltration in the cortex. Bogolomez [4] demonstrated that degenerative changes developed in the suprarenal cortices of cats given injections of diphtheria toxin alone, while the cortical cells of cats given injections of toxin-antitoxin were normal. The cortices of the suprarenal glands of children with scarlet fever, acute and chronic osteomyelitis, staphylococcic septicemia and other infections, examined post mortem, showed many pathologic changes (Thomas [5]). It was explained that as the cells of the suprarenal cortex were involved in the defense mechanism, they soon degenerated as a result of the great strain placed on them by massive

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3. Oppenheim, R., and Loeper, M.: Lésions des capsules surrénales dans quelques infections expérimentales aiguës, Arch. de méd. expér. et d'anat. path. **13**:332, 1901.

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5. Thomas, E.: Ueber die Nebenniere des Kindes und ihre Veränderungen bei Infectionskrankheiten, Beitr. z. path. Anat. u. z. allg. Path. **50**:283, 1911.

inoculations of pathogenic bacteria. Death was believed to be due, in part, to suprarenal insufficiency.

Removal of the suprarenal glands is attended by a reduction in the resistance of the animal to toxins and infection. Rats are normally resistant to diphtheria toxin, but after suprarenalectomy they lose their natural immunity (Oppenheim and Loeper [6]). Lewis [7] demonstrated that rats become more sensitive to cobra venom and epinephrine after suprarenalectomy. Scott [8] reported that the decreased resistance of suprarenalectomized rats to killed streptococci and staphylococci was probably due to a lack of suprarenal cortex. It was later shown by Blanchard [9] that the opsonic power of the serum of suprarenalectomized cats was greatly decreased.

With the recent development of many methods for making potent extracts of the suprarenal cortex, interest has again been aroused in the relationship between the suprarenal cortex and resistance to infection. Scott and Bradford [10] found that the extract (Swingle and Pfiffner [11]) greatly increased the resistance of suprarenalectomized rats to killed typhoid bacilli. Hartman and Scott [12] reported that the ether-alcohol extract of Hartman [13] raised the resistance of suprarenalectomized rats to bacterial intoxication. Whitehead and Smith [14] injected cortex extract intramuscularly into hospitalized patients with bacterial intoxication and reported favorable results. In view of these and other investigations, it was deemed advisable to study the effect of extract of the suprarenal cortex on experimentally produced maxillary sinusitis.

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12. Hartman, F. A., and Scott, W. J. M.: Protection of Adrenalectomized Animals Against Bacterial Intoxication, Proc. Soc. Exper. Biol. & Med. **28**:478, 1931.

13. Hartman, F. A.: Cortin, Vital Hormone of the Adrenal Cortex, Endocrinology **14**:229, 1930.

14. Whitehead, R. W., and Smith, C.: Effect of Adrenal Cortex Extract on the Course of Certain Human Infections, Proc. Soc. Exper. Biol. & Med. **29**:672, 1932.

MATERIAL AND METHODS

A portion of the extract of the suprarenal cortex used in these experiments was supplied by Dr. R. L. Zwemer, and the rest was made in this laboratory according to the improved method of extraction given by Zwemer, Agate and Schroeder,[15] which briefly is as follows: Five kilograms of whole beef suprarenal gland is ground in a food chopper and mixed with 3.5 Kg. of anhydrous sodium sulphate, to which is added 7.5 liters of benzene; the whole is stirred for twelve hours and strained through cheesecloth. The suprarenal-sulphate residue is again extracted with 7.5 liters of benzene for four hours and strained. The two benzene extractions are combined, filtered and concentrated in vacuo to 2 liters, to which is added 1 liter of two-tenths normal hydrochloric acid; the whole is shaken and allowed to settle. The supernatant benzene is drawn off and shaken with another liter of two-tenths normal hydrochloric acid. The hydrochloric acid emulsions are combined; benzene is removed in vacuo, which breaks up the hydrochloric acid-fat emulsion. The fat separates out and is removed by straining and filtering. The clear yellow acid extract is purified by shaking against 1 liter of benzene and simultaneously adding saturated sodium bicarbonate to a p_H of about 8. The benzene layer is saved. The aqueous layer is shaken with another liter of benzene. The two benzene fractions are combined and concentrated to 50 cc. and shaken with two successive fractions of 50 cc. of two-tenths normal hydrochloric acid, which are combined; the benzene is completely removed in vacuo, and the extract is filtered through a Berkefeld filter and sealed in sterile ampules. The acid extract is neutralized with two-tenths normal sodium hydroxide before using.

Maxillary sinusitis was produced in fifty-two rabbits by irrigating the sinuses every forty-eight hours for ten days with from 0.3 to 0.5 per cent solution of sodium alizarin sulphonate. Previously I [16] had shown that irrigating the maxillary sinuses with aqueous solutions of alizarin and other calcium-combining substances results in the cessation of ciliary movement and ultimate disappearance of the cilia. Eosinophilic infiltration takes place within ten days after the first irrigation. With the loss of ciliary movement, stagnation and infection, as evidenced by the purulent nasal discharge, result within ten days following the last irrigation with alizarin. When a nasal discharge appeared, the rabbits were divided into two groups. One was given three intramuscular injections of the extract of the suprarenal cortex in doses of 0.5 cc. per kilogram of body weight over a period of one week. The second or control group received no extract. Both control and experimental animals were killed seventeen days after the last sinus irrigation. The mucosa was removed from the lateral and medial walls of the maxillary sinus and fixed in formaldehyde solution. Paraffin sections 4 microns in thickness were prepared and stained with hematoxylin-eosin and Giemsa stains.

RESULTS

When examined grossly, the sinuses of the rabbits treated with extract of the suprarenal cortex showed no pus. Microscopic examination revealed no evidence of infection except in one rabbit, which

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showed a slight infiltration of neutrophils and eosinophils into the basement membrane. The ciliated columnar epithelium was normal in all the treated rabbits. The sinuses of the rabbits used as controls contained varying amounts of macroscopic pus. Microscopically, the

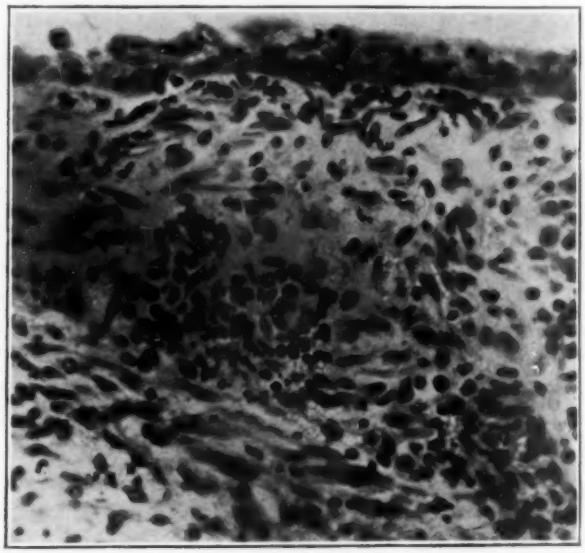

Fig. 1.—The sinus mucosa of a rabbit seventeen days after the last irrigation with 0.3 per cent alizarin. The mucosa shows marked thickening of the basement membrane with infiltration by polymorphonuclear leukocytes and lymphocytes. The epithelium is flattened, approaching the squamous type; cilia are absent.

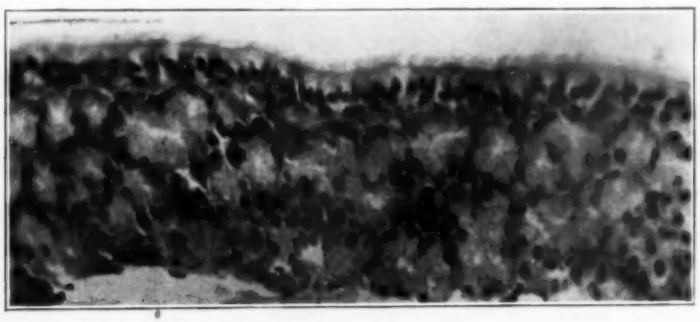

Fig. 2 (rabbit 216).—Ten days after the last sinus irrigation with 0.3 per cent alizarin, the rabbit received three intramuscular injections of extract of the suprarenal cortex over a period of one week. The sinus mucosa shows no evidence of infection.

basement membrane was greatly thickened and infiltrated by polymorphonuclear leukocytes and lymphocytes. The epithelium showed loss of cilia and marked flattening, closely resembling squamous cells.

The suprarenal glands are apparently necessary for the maintenance of water balance. Zwemer [17] and Swingle and his associates [18] observed that suprarenalectomy leads to dehydration, as shown by the rapid loss in weight, the loss in elasticity of the skin, the dry mucous and serous membranes and the decreased peripheral circulation. The red blood cell count, hematocrit readings and viscosity of the blood show great increases over the normal figures. Along with the loss of water there are a diminished alkali reserve (Swingle and Eisenman,[19] Yonkman [20]), impairment of renal function and cloudy swelling of the epithelium of the renal tubules (MacMahon and Zwemer [21]).

When extract of the suprarenal cortex is injected intramuscularly into suprarenalectomized animals, the volume of the plasma and the carbon dioxide capacity of the blood increase in from twenty-four to seventy-two hours. Zwemer [22] reported that cortical extract increases the carbon dioxide capacity of the blood of normal cats and rabbits about 26 per cent. However, he found that the rise in the carbon dioxide capacity was not uniform. In animals with an initial high carbon dioxide capacity of the blood this capacity decreased, while in those with an initial low carbon dioxide capacity of the blood, the capacity increased. Figures on the carbon dioxide content of the blood plasma of the animals used in this experiment show that the greatest change occurs about seventy-two hours after the injection of the cortical extract. The most pronounced change takes place in the animals with an initial low figure (table 1), while those with an initial high carbon dioxide capacity show a slight change or a decrease. Table 2 gives the data on the effect of cortical extract on the hemoglobin, p_H and carbon dioxide content of the blood of rabbits with sinusitis before and seventy-two hours after receiving extract of the suprarenal cortex. The hemoglobin (Newcomer method) was consistently reduced except in animal

17. Zwemer, R. L.: An Experimental Study of the Adrenal Cortex: II. Prolongation of Life After Complete Epinephrectomy, Am. J. Physiol. **79**:658, 1927.

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233, which showed no change (the p_H and carbon dioxide content were unaffected). The inconsistent findings on the carbon dioxide content can probably be ascribed to the degree of dilution of the blood. An increase in the volume of the plasma with an increased flow of blood

TABLE 1.—*Effect of Extract of Suprarenal Cortex on Carbon Dioxide Content of Rabbit's Blood* *

Rabbit	Normal at Time of First Injection	48 Hours After First Injection	72 Hours After First Injection; Second Injection Given	24 Hours After Second Injection	72 Hours After Second Injection
201	48.9	45.1	50.7	52.0	53.6
202	37.0	39.1	49.6	48.4	34.3
203	34.4	44.1	45.0	48.1	55.7
204	58.4	36.6	52.0	58.4	53.9
211	54.3	....	62.5	47.8	48.1
212	60.6	....	48.5	52.8	54.8
213	34.2	....	42.9	36.7	39.9
214	38.8	....	47.8	48.6	49.3
215	38.1	....	41.6	39.7	47.0
216	43.9	....	48.9	54.8	58.2

* The extract was given in doses of 0.5 cc. per kilogram of body weight. The carbon dioxide is given in per cent by volume.

TABLE 2.—*Effect of Extract of Suprarenal Cortex on Hemoglobin, p_H and Carbon Dioxide Content of Rabbit's Blood*

Rabbit	Hemoglobin, Gm.	p_H	Carbon Dioxide Content, per Cent by Volume	Comment
228	12.3	7.46	41.4	Normal
	9.9	7.51	50.9	72 hours after injection of 0.5 cc. per kilogram
229	10.2	7.41	39.1	Normal
	7.8	7.56	46.0	72 hours after injection
230	11.0	7.39	47.0	Normal
	6.9	7.47	43.4	72 hours after injection
231	10.3	7.45	55.0	Normal
	5.8	7.38	31.6	72 hours after injection
232	9.2	7.43	42.4	Normal
	7.3	7.46	48.6	72 hours after injection
233	9.6	7.41	39.3	Normal
	9.3	7.40	41.9	72 hours after injection
234	10.1	7.46	45.9	Normal
	8.3	7.39	39.1	72 hours after injection
235	11.4	7.38	35.4	Normal
	7.8	7.43	44.6	72 hours after injection

favors a more efficient exchange of blood gases up to a certain point; then the relative decrease in hemoglobin with a diminished oxygen-carrying power per unit volume of blood (anoxemia) neutralizes or counteracts the original beneficial effect of the increased rate of blood flow. The variation in the response of each animal to the same amount of extract per kilogram of body weight cannot be explained.

Microscopic examination of the suprarenal glands of rabbits with sinusitis showed no pathologic changes. The zona glomerulosa, zona fasciculata and the inner or reticular zone of the cortex showed no evidence of inflammatory reaction. The cells contained the normal amount of vacuoles (fig. 3). Cortical extract produced a marked increase in the number and size of the lipid granules in the cytoplasm of the cells of the cortex (fig. 4). This increase in lipid granules (seen as vacuoles in paraffin sections) was probably the result of increased activity of the cells of the cortex. Aside from the highly vacuolated cytoplasm, no other changes, such as hyperemia, cellular infiltration and degenerative changes, were noted.

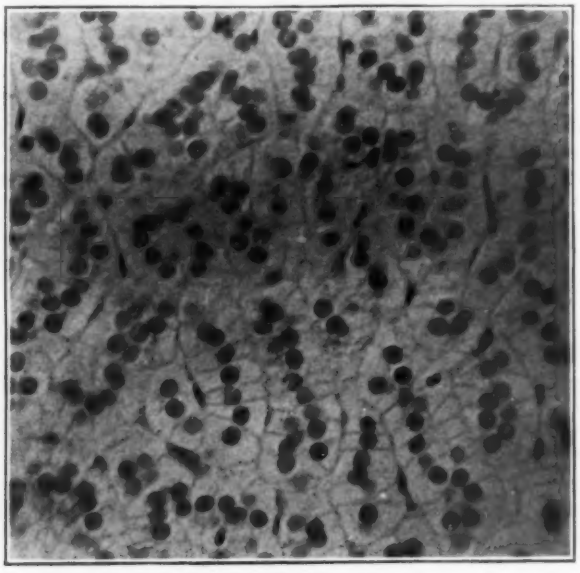

Fig. 3.—The cortical cells of the suprarenal gland of a normal rabbit.

The results of these experiments on uncomplicated experimentally produced sinusitis indicate clearly that the extract of the suprarenal cortex exerts a marked influence on the resistance of the organism to infection. The exact mechanism of this response has not been determined. No noticeable change in either the total or the differential white cell count occurred. White cell counts made on normal and on experimental rabbits at the same hour of the day showed a wide variation from day to day, so that it was practically impossible to observe any change that might be attributed to the action of the extract.

The chemical picture of the blood in suprarenal insufficiency in experimental animals and that of dehydration resulting from nutritional

disturbances in infants have been found to be similar (Sullivan, MacLean and Zwemer [23]). With the loss of water, fixed bases and chloride anions in the stool and vomitus, dehydration and acidosis or alkalosis result. The viscosity of the blood and the red blood cell count increase. The subsequent oliguria produces retention of nitrogen. Extract of the suprarenal cortex increases the absorption of water from the gastrointestinal tract and of water administered parenterally, and restores a normal water balance in the blood and tissues.

The decrease in the phagocytic power of the leukocytes in suprarenal insufficiency may be due to the decreased peripheral circulation

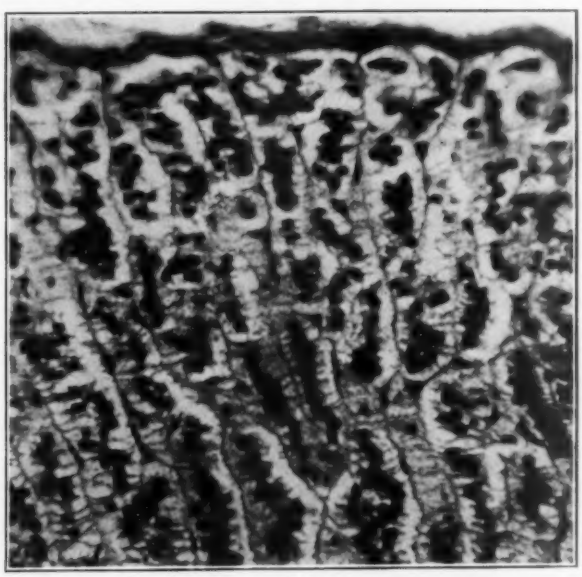

Fig. 4.—The cortex of the suprarenal gland of a rabbit twenty-four hours after the intramuscular injection of 0.5 cc. per kilogram of body weight of extract of the suprarenal cortex. The cortical cells are highly vacuolated.

and concomitant disturbances in the nutrition of the tissues. The administration of the hormone of the suprarenal cortex increases the volume of the plasma and the rate of the flow of blood not only in patients with suprarenal insufficiency, but also in normal persons. It is probable that with an improvement in the nutrition of the various organs and organ systems the protective mechanism of the body becomes more efficient.

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23. Sullivan, R. C.; MacLean, A. B., and Zwemer, R. L.: A Comparison of the Chemical Composition of the Blood in Acute Intestinal Intoxication and Experimental Adrenal Insufficiency in Animals, Am. J. Dis. Child. **43**:1279 (May, pt. 1) 1932.

The extract of the suprarenal cortex has been used in the treatment of acute and chronic sinusitis, retrotonsillar abscesses and other infections about the ear, nose and throat. The results are interesting. The number of patients treated does not permit evaluation of the action of the extract at this time.

SUMMARY

Rabbits with experimentally produced maxillary sinusitis were successfully treated with an extract of the suprarenal cortex. After three intramuscular injections of the extract, the rabbits showed no evidence of infection of the sinus mucosa on microscopic examination. The extract of the suprarenal cortex produced a consistent decrease in hemoglobin and a change in the alkali reserve.

After the administration of the cortical extract, the cells of the suprarenal cortex showed an increased number of vacuoles in the cytoplasm.

NASAL SECRETIONS

THE VALUE OF CYTOLOGIC EXAMINATION TO THE RHINOLOGIST

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AND
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Attention has been drawn in recent years to the value of the cytologic examination of nasal secretions in the diagnosis of diseases of the nose and accessory sinuses. Sewall [1] stressed the presence of polymorphonuclear leukocytes in chronic sinusitis; Hansel,[2] Kahn and Stout,[3] Johnson and Goldstein [4] and others noted the importance of eosinophils in the diagnosis of nasal allergy. We wish to present the results of a hundred routine examinations of nasal smears taken from patients with acute and chronic rhinitis and sinusitis, and to emphasize the value of cytologic examination to the rhinologist.

Results of Investigation in a Hundred Cases

	Number of Cases Showing Given Percentage of Eosinophils in Nasal Smear				
Condition	0	1-5%	5-10%	10-100%	Total
Acute infections of upper respiratory tract.....	11	0	0	0	11
Vasomotor rhinitis* (with and without superadded infection)	2†	0	9	58	69
Chronic suppurative sinusitis.....	18	2	0	0	20
					100

* In an investigation of the sixty-nine cases of vasomotor rhinitis for allergy, skin tests gave the following results: positive, 31; negative, 9; incomplete, 29.
† The two cases of vasomotor rhinitis in which no eosinophils were shown in the nasal smear were cases of fall hay fever seen during the quiescent period.

Smears were made by having the patient blow material from the nose onto a glass slide. This material was then spread as one spreads a blood smear. The smears were allowed to dry in the air and later

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2. Hansel, F. K.: Clinical and Histopathologic Studies of the Nose and Sinuses in Allergy, J. Allergy **1**:43, 1929.

3. Kahn, I. S., and Stout, B. F.: Allergy and Infection, J. A. M. A. **99**:1494 (Oct. 29) 1932.

4. Johnson, M. C., and Goldstein, D. W.: Allergy and the Cytologic Examination of Nasal Smears, Arch. Otolaryng. **16**:808 (Dec.) 1932.

were stained with Wright's stain. It was our practice rather to overstain at first and then to differentiate in methyl alcohol, as by this means the eosinophilic granules are well seen. This differentiation is important in the preparation of a good slide, because if the staining is a little deep, it is almost impossible to see granules in the eosinophils, and these cells then resemble mononuclear cells and may be mistaken for small lymphocytes. The percentages quoted in the table refer only to eosinophils and polymorphonuclear neutrophils. In order better to study the other cells in the smears, many were fixed in Zenker's solution to which a dilute solution of formaldehyde had been added, and stained with Maximow's hematoxylin-eosin-azur. By this stain various epithelial cells, cubical, squamous and mucus-secreting types, were well seen, and occasional lymphocytes were found. The lymphocytes were by no means numerous; we rarely saw more than from 6 to 10 in a smear. This observation does not agree with the findings of Johnson, who reported as many as 100 per cent lymphocytes in some cases. It was found that when the secretion was very watery there were fewer cells, and that the greatest number of cells were found in clumps of mucus.

There are various points of interest which appealed to us in this study and which we think are worthy of emphasis. In the series there were no fewer than nine cases (14.9 per cent) showing an eosinophilia in the nasal smear of from 5 to 100 per cent and no positive cutaneous reactions. In only one of these cases was the eosinophil count less than 50 per cent. In each case a thorough and complete allergic investigation was carried out in the allergy clinic by Dr. Harry L. Huber. The absence of demonstrable allergy in the presence of a high eosinophil count in the nasal smear does not agree with the findings of Kahn [3] or of Johnson.[4] Such cases seem to fit in with the so-called "nonallergic" [5] or "reflex" [6] type of vasomotor rhinitis, although we cannot dismiss the possibility that the patients were tested during a "refractory" period, thus accounting for the absence of positive reactions of the skin.

We believe, however, that the real value of routine cytologic examination of the nasal secretions lies in the indication it gives of the presence of vasomotor rhinitis as the basis of chronic suppurative sinusitis. When a chronic infection of the sinuses supervenes in vasomotor rhinitis, the clinical signs of the vasomotor rhinitis are frequently masked by those of the superimposed infection. It is in such cases that the results of surgical procedures alone have proved so unsatisfactory. These unsatisfactory results not only are a disappointment to the rhinologist, but have led to the belief among the laity that "sinusitis cannot be cured," and even to prejudice among members of the medical

5. Walsh, T. E.: Vasomotor Rhinitis, Arch. Otolaryng. **16**:83 (July) 1932.
6. Hansel, F. K.: Vasomotor Rhinitis, J. A. M. A. **82**:15 (Jan. 5) 1924.

profession against intranasal surgical procedures. That suitable surgical treatment is essential in most cases of chronic suppurative sinusitis cannot be gainsaid; but it is still more certain that the recognition and proper investigation and treatment of complicating vasomotor rhinitis are even more essential for success.

The ratio of eosinophils to neutrophils is definitely lowered in the presence of infection superimposed on vasomotor rhinitis. This was well shown in the case of C. S., who was first seen with an acute infection of the upper respiratory tract (common cold). The routine smear showed 50 per cent eosinophils. She was seen again sixteen days later, after the cold had cleared up, and at that time a smear showed that all the cells were eosinophils. On the contrary, in the cases of uncomplicated sinusitis, either acute or chronic, in our series, the nasal smear never contained more than 1 per cent of eosinophils; they contained, however, an abundance of polymorphonuclear neutrophils. The normal physiologic nasal secretion, such as that obtained from the "running nose" of a person outdoors on a cold day, contains very few cells; perhaps half a dozen or so may be found by assiduous search through a slide. In pathologic secretions, however, the cells are numerous, with the exception of secretions taken in the early stages of an acute cold or those of acute hay fever. In the former the cells are polymorphonuclear neutrophils; in the latter, eosinophils.

It is of interest to note that the eosinophilia in the nasal secretions bears no constant relation to that in the blood. Brown [7] noted that the eosinophilia in the sputum in cases of asthma runs exactly parallel to that in the blood. In vasomotor rhinitis we have found that although the nasal secretion may contain large numbers of eosinophils, the eosinophilia in the blood is frequently only 1 or 2 per cent, while in other cases it may range as high as 14 per cent.

SUMMARY

Normal nasal secretions contain few cells. The secretions in pathologic nasal conditions contain an abundance of cells.

The presence of eosinophils in nasal secretions is indicative of vasomotor rhinitis. No allergy was demonstrable in 14.9 per cent of the cases in which there was a high percentage of eosinophils in the nasal secretions.

When vasomotor rhinitis was complicated by infection the ratio of eosinophils to neutrophils was lowered, but in cases of infection without vasomotor rhinitis the eosinophil count in the nasal secretion was never higher than 1 per cent.

7. Brown, G. T.: Eosinophilia in Asthma, Hay Fever, and Allied Conditions, J. Lab. & Clin. Med. **12**:1145, 1927.

In cases of allergic rhinitis, eosinophils were absent from the nasal secretion during quiescent periods.

The number of eosinophils in the nasal secretions bore no constant relation to that in the blood.

Routine examination of smears of nasal secretions affords a simple and effective laboratory aid in the diagnosis of vasomotor rhinitis when the clinical signs of the condition are masked by those of a complicating infection.

A LATERAL HEAD-LOW POSITION FOR NASAL AND SINUS TREATMENT

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Specific methods of treatment of the more common nasal and sinus conditions are seldom discussed in the current literature. Yet such conditions are the ones that really demand most of an otolaryngologist's attention. A remark or so on the head-low position as a means of conservative treatment may, therefore, not be out of order. In my opinion, this method has greater possibilities of usefulness than are accorded it at present, and is often productive of such gratifying results that it seems worthy of more general application in practice.

This seems especially true since the description by Proetz[1] of a method whereby fluid can be made to enter all the sinuses at once by suction displacement of the contained air. Such fluid may then remain for a considerable time in direct contact with the sinus mucosa. In principle, the head-low position offers a rational method of obtaining prolonged contact of a desirable drug with the mucosa of the upper areas of the nasal chambers. To me, it seems distinctly superior in most instances to the widely employed nasal tampons, because of the greater surface area reached, and the longer symptomatic relief.

The head-low position generally known and used is that of exaggerated extension of the head, as described by Proetz,[1] wherein the patient lies supine with his head hanging over the end of the cot. In order that fluid placed in the nose will not pass directly into the nasopharynx, extreme extension of the head is here required. Sufficient extension is uncomfortable for the average patient and is nearly impossible for persons with short necks. Also, on recovering from this position, it is difficult to avoid having some of the fluid enter the pharynx.

All the sinus ostea lie in the upper half of the nasal fossae and many in the upper posterior third. Logically, then, this is the area one should attempt to reach. In order to flood this region properly with a treatment solution, the head must be placed with the cribriform plate low and horizontal. To accomplish this, I should like to call attention to a lateral head-low position. This position I have found to be effective, generally applicable and relatively comfortable. It is quite possible that others also may find use for it in certain cases.

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1. Proetz, Arthur W.: Displacement Irrigation of Nasal Sinuses, Arch. Otolaryng. **4**:1 (July) 1926; Further Data on the Displacement Method in Sinuses, Ann. Otol., Rhin. & Laryng. **36**:297 (June) 1927.

The patient is seated on a cot immediately adjacent to a stack of two or three pillows. He is then bent laterally on the pillows, the lower arm being kept at the side so as to rest beneath him, with the elbow flexed. With the body in a strictly sidewise position, the head is now bent downward (laterally) to as nearly up-side-down as possible. It is necessary that the lower shoulder be kept on the pillows, otherwise the tendency of the patient is to override until his shoulder rests on the cot. The ability to approach a perfectly vertical position of the head is about in proportion to the length of the patient's neck, but in any event it will be found that the olfactory area is down and level. Fluid placed in the nostrils now will readily gravitate to the superior nasal areas. The patient is instructed to breathe only through the mouth in order to avoid snuffing any fluid into the pharynx. If treatment or displacement is especially desired in the posterior ethmoid-sphenoid region, the face may be turned slightly upward, thus converting this area into a shallow fossa.[1] Patients find this position relatively comfortable and are able to hold it several minutes without complaint. Before the patient arises, his face is first turned downward, allowing whatever fluid is in the nose to run out of the nostrils. It is unnecessary that the patient at any time get the slightest taste of the drug used. Small children may be turned laterally and, if necessary, the head held in the lap or between the knees. A few elderly plethoric persons seem unable to tolerate easily any head-low position.

This lateral position I have found especially useful in acute and subacute infections of the frontal and associated ethmoid sinuses. Here it is desirable to shrink and drain a diseased area with the least possible trauma and reaction. By this procedure no intranasal instrumentation is necessary, and there is usually prompt and gratifying symptomatic relief of many hours' duration. From three to five minutes seems to be ample time for treatment. Of the several preparations that might be used, my decided preference is 1 per cent ephedrine in physiologic solution of sodium chloride, warmed. This gives a minimum of discomfort (practically none) when instilled in the nose, and disquieting after-effects have not been observed. By the Proetz displacement method, the fluid can be made actually to enter the sinuses if desired.

During the past year and a half this procedure has been used also in an attempt to produce clinical improvement, in selected cases, through efforts at local immunization of the nasal and sinus membranes to the specific offending organisms. In this work bacteriophage-lysed [2] sterile antigen solutions are used. These have been prepared from autogenous nasal cultures by Albert Krueger of the bacteriologic department of the University of California Medical School.

In conclusion, for those who use iodized oil as a contrast medium in diagnosis, this procedure will be found to be of use in the examination of ethmoids and sphenoids by the displacement method as described by Proetz.

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2. Northrup, J. H., and Krueger, A. P.: J. Gen. Physiol. **15**:329, 1931.

Clinical Notes

BRONCHOPULMONARY CATHETERS FOR INTRAPULMONARY THERAPY

M. J. Mandelbaum, M.D., New York

During the past few years an awakened interest has been noted in direct intrapulmonary therapy by means of cannulated instruments, particularly the various types of nonmetallic bronchial catheters.

Ten years ago I devised a styletted, soft rubber catheter,[1] made by the George P. Pilling & Son Company of Philadelphia, which I have been using since that time. This was originally intended for use in making endobronchial instillations of iodized poppy seed oil 40 per cent for bronchographic purposes.

Others had devised similar instruments before I called attention to their use, and have done so since. The bronchial catheter was first suggested to Horace Green [2] of New York by Marshall Hall of London in 1855. Darter,[3] in 1893, was the first to utilize a styletted catheter for this purpose, so far as a careful search of the literature reveals.

Following my use of the instrument, Stitt,[4] in 1927, employed a catheter. Garcia Vicente,[5] in 1928, Oughterson,[6] in 1931, and de Pablo,[7] in 1932, devised other variations either in the catheter itself or in the instruments used in its introduction into the trachea.

Lewy [8] recently described another means of introducing a bronchial catheter. He uses a waxed linen ureteral catheter of large caliber through a Mosher metal "life-saver" with the tip cut off. According to his article, he uses it for suction only, to relieve bronchial hypersecretion by aspiration.

Several years ago Pitman,[9] of New York, exhibited a metal cannula of similar construction which permitted the introduction of a tracheal catheter by means of

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1. Mandelbaum, M. J.: Bronchoclysis: An Original Method for Prolonged Intrapulmonary Therapy, Arch. Phys. Therapy **18**:673 (Nov.) 1932.

2. Green, Horace: On Injection of the Bronchial Tubes and Tubercular Cavities of the Lung, Am. M. Month. **3**:5 (Jan.) 1855.

3. Darter, J. M.: Laryngeal Catheter, Texas Cour.-Rec. Med. **10**:41, 1893.

4. Stitt, H. L.: Rubber Catheter for a Bronchial Tube, Tr. Am. Acad. Ophth. 1927, p. 369.

5. Garcia Vicente, S.: Injections into Trachea, Simplified Technic, Progresos de la clin. **36**:457 (July) 1928.

6. Oughterson, A.: A New Forceps for the Intrabronchial Injection of Iodized Oil, Arch. Otolaryng. **14**:617 (Nov.) 1931.

7. de Pablo, V.: Simple Technic for Intrapulmonary Therapy: A Modification of Garcia Vicente's Method, Siglo méd. **89**:49 (Jan. 16) 1932.

8. Lewy, Alfred: Aspiration of Fluid from the Lung: An Improved Method, Arch. Otolaryng. **17**:86 (Jan.) 1933.

9. Pitman, Louis: Personal communication.

a small attached ratchet which propelled the catheter, slowly and easily, a little at a time, to the desired region. Lewy's instrument is practically of the same type, except for the ratchet.

In 1925 I began experimentation with longer catheters, particularly the fine-calibered pulmonary catheters of ureteral type. In 1932, in a paper on bronchoclysis,[1] I described in detail the uses of these types of catheters.

The great advantage of the styletted catheter for tracheal intubation is that the distal end of the catheter may be quickly introduced into the desired region, the stylet removed and the soft catheter left in situ. This permits retention of the catheter for prolonged periods without distress.

The stiff metallic tubes used for introducing the catheters must be left in place while the soft catheters are being used, and, not being flexible, they are incapable of remaining in position for prolonged periods.

The styletted catheter (fig. 1) has none of the disadvantages of the other types and besides possesses additional advantages.

After my investigations with the styletted catheter, I made a further study of the possibilities of endobronchial catheters.

For the purpose of bronchoclysis, the ureteral type of pulmonary catheter is superior to the urethral forms, since if it is of radiopaque material, the distal end may be introduced, under fluoroscopic guidance, into the smallest bronchi.

Fig. 1.—The styletted catheter.

By a combination of various curves, I have devised a series of small-calibered catheters which can be introduced into any lobe or lobule of either lung, permitting the instillation of medicaments directly into the affected area as well as prolonged retention of the instrument.

The fine-calibered catheter is particularly valuable for the bronchial drip, or bronchoclysis, in which isotonic aqueous remedial agents are slowly introduced, drop by drop, at a rate conforming to the rapidity of the remarkably active absorption of the rich pulmonary lymphatic system.

The fact that the fluids are instilled at a rate commensurate with the absorptive power of the bronchopulmonary tissue prevents the accumulation of excessive fluid (lymphatic infiltration) in the parenchyma. When the procedure is properly performed, it is impossible to detect any shadows or other abnormal markings in the lungs, by either roentgen or physical examination, during or subsequent to the injection, the fluid being absorbed as rapidly as it is introduced.

Direct endobronchial therapy may be applied (1) transnasally, (2) transorally, (3) transtracheally and (4) transthoracically. Each of these routes requires a special instrumentarium and a different technic.

The finger holder for the laryngoscopic mirror (fig. 2) facilitates the introduction of the bronchial catheter by enabling the operator to hold the mirror and the patient's tongue with one hand. This makes it possible to dispense with the services of an assistant, but its chief advantage is in permitting coordination between the movements of the muscles of the patient's throat and tongue and the movements of the operator's mirror-holding hand.

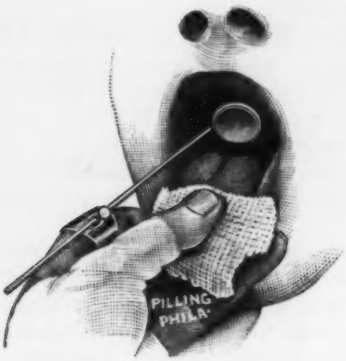

Fig. 2.—The finger holder for the laryngoscopic mirror.

Fig. 3.—The laryngeal catheter-introducing forceps.

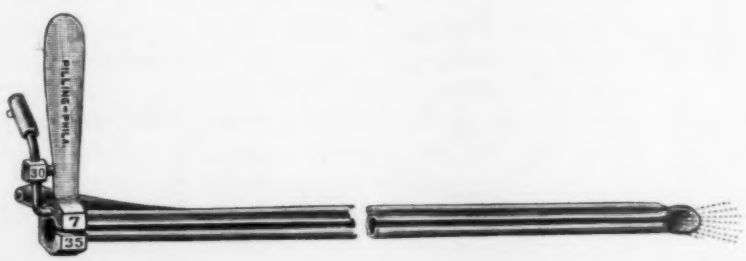

Fig. 4.—The slotted bronchoscope for introducing pulmonary catheters into definitely selected bronchi.

A complete description of the preparation of the patient, including anesthesia and technic, has been published.[1]

The soft catheters require instruments of various types for their introduction supralaryngeally: a stylet, as devised by Darter[3] and by me,[1] a forceps, as devised by Oughterson,[6] or a special laryngeal speculum, such as that designed by de Pablo.[7] I introduce soft rubber catheters either with a stylet or by means of a special catheter-introducing forceps (fig. 3).

The stiffer, though flexible, silk or waxed linen catheters may also be introduced by these means, but in trained hands they can be used without special introducing instruments.

While Lewy[8] uses the waxed linen catheter for aspiration only, I utilize it, in addition, for introducing endobronchial fluid medication following the removal by suction of excessive secretions in nonbronchoscopic direct intrapulmonary therapy.

I have also devised ureteral types of waxed linen pulmonary catheters for use bronchoscopically, employing a special "slotted" bronchoscope (fig. 4), also made by the George P. Pilling & Son Company, which is easily withdrawn, leaving the catheter in place.

I feel it important to emphasize that no type of endobronchial therapy should be attempted without a full appreciation of the anatomy, physiology and pathology of the respiratory system, and then only after careful preliminary diagnostic study and preparation of the patient, as previously described.[1]

It is well to mention that endobronchial therapy should at all times be considered merely an adjuvant to bronchoscopy and bronchoscopic therapy, as the indiscriminate use of nonbronchoscopic endobronchial therapy without proper preliminary clinical study and bronchoscopic investigation will sooner or later lead to grief.

The future will undoubtedly witness a much wider adoption of this valuable addition of the pulmonary catheter in both the bronchoscopic and the nonbronchoscopic therapeutic armamentarium for pulmonary diseases.

27 West Seventy-Second Street.

Progress in Otolaryngology

A Summary of the Bibliographic Material Available in the Field of Otolaryngology

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CHRONIC PROGRESSIVE DEAFNESS, INCLUDING OTOSCLEROSIS AND DISEASES OF THE INNER EAR

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OTTAWA, CANADA

Albrecht,[1] in a paper on the constitutional factor of otosclerosis, expressed the view that heredity is the underlying cause. This is hard to prove, as the condition is usually a recessive characteristic and may pass over several generations without showing itself, and some cases apparently occur sporadically. As has been pointed out by others, it is difficult to follow the disease through generations in which the individuals are scattered and to be sure that those who are deaf are really suffering from otosclerosis. On the other hand, many people have the characteristic bony changes of otosclerosis without deafness.

Albrecht does not believe that otosclerosis is caused by endocrine disturbances, but thinks that the labyrinthine changes may be related to them. The theory that otosclerosis is caused by chronic irritation of the bone has been supported by Manasse, Lange, O. Mayer, Nager and others. The constitutional factor is as important as the irritation, whether the latter is from endocrine disturbance, mechanical strain (Brühl, O. Mayer) or chronic hyperemia (Wittmaack). These local factors can produce otosclerosis only when added to an inherently weak bone from faulty heredity. Albrecht stated that the development of otosclerosis on a foundation of bony malformation is possible, but that its development on a basis of chronic irritation is much more probable. These hypotheses form the nearest approach to an explanation of the simultaneous involvement of the nervous and bony elements.

Davenport,[2] in a report to the American Otological Society on the genetic study of otosclerosis, emphasized the difficulties in obtaining reliable data owing to uncertainty of diagnosis, etc. He studied and analyzed, in all, 470 pedigrees collected from the literature and from reports by his field workers. His conclusions are in part as follows:

The hypothesis of two dominant genes is not negatived in any of the groups of matings. The hypothesis that otosclerosis depends on a single dominant factor, a

1. Albrecht: Ztschr. f. Hals-, Nasen- u. Ohrenh. **29**:18, 1931.
2. Davenport: Tr. Am. Otol. Soc. **22**:50, 1932.

single recessive factor, a sex-linked recessive and dominant autosomal gene, two recessive genes (one sex-linked), also on a dominant and a recessive autosomal gene, all meet with difficulties in application. The conclusion is reached that the simplest formula that approaches agreement with the findings is that of the two dominant genes, of which one is sex-linked.

Gottlieb [3] quoted Hammerschlag, who maintained that congenital deafness and otosclerosis were only differing forms of the same degenerative process. He gave as examples the cases of two families in which congenital deafness and otosclerosis occurred side by side. Furthermore, otosclerotic changes in the bone have not infrequently been found in congenital deaf-mutes.

In a review of the literature Gottlieb produced a great deal of evidence to show that heredity is the main factor in progressive deafness, whether it acts through vascular instability or by other means. Most cases of progressive deafness show stigmas of degeneration.

The symptom of a degenerative disposition demonstrable in all cases of otosclerosis is the lability of the circulation, which has its cause in the hyperirritability of the vasomotor nerves and in the lability of the heart action.

He concluded with the following sentences:

In every case of nonsuppurative bilateral deafness in which there is no local pathologic defect other general objective and subjective abnormalities occur which when correlated lead the way to finding their cause as well as that of the deafness. From this standpoint, I do not hesitate to say that constitutional deafness is a preventable and curable condition within certain well defined limits. It can be prevented by the institution of yearly tests with the audiometer of all persons above puberty. A drop in hearing below the original level should be immediately followed by a complete subjective and objective survey, and all abnormalities should be corrected if possible. Hardness of hearing is curable in the sense that in the early cases prompt response to treatment is the rule. In cases that have existed over five years complete recovery is not to be expected, although some improvement does occur or the progress of decline in hearing is definitely checked.

Werner,[4] an enthusiastic pupil of Wittmaack, summarized the evidence in favor of Wittmaack's theory of venous congestion as the cause of otosclerosis. No relevant data were brought forward that have not already been mentioned in these pages.

E. P. Fowler, Jr.[5] presented in a clear form the main points in four of the well known theories of the cause of otosclerosis. Brühl's theory is that otosclerosis is a constitutional hereditary condition and not a disease. It is a condition in which the old bone is absorbed and replaced by new spongy bone, where it is subjected to constant strain from the

3. Gottlieb, M. J.: Constitutional Deafness, Arch. Otolaryng. **16**:814 (Dec.) 1932.

4. Werner, C. F.: Internat. Zentralbl. f. Ohrenh. **35**:141, 1932.

5. Fowler, E. P., Jr.: Ann. Otol., Rhin. & Laryng. **41**:175, 1932.

pull of the annular ligament on the one hand and that of the tensor tympani muscle acting through the processus cochleariformis on the other.

Wittmaack's theory is that otosclerosis is a unique pathologic condition brought about by venous stasis. He noted the large size of the veins in the foci and concluded that this is the cause of the changes in the bone. He claims to have produced identical lesions in the labyrinth of the fowl by causing obstruction of the efferent veins. (Many objections have been raised to the value of the results so obtained, such as the difference in form and structure between the avian labyrinth and that of man. No comparable gross interruption of the venous return has been seen in man. So far there has been complete failure to reproduce lesions in mammals.—*Abstractor.*)

M. Weber, who has made extensive studies of the growth and pathology of bone, believes that otosclerosis is identical with a localized form of osteitis fibrosa.

O. Mayer's theory has some resemblance to Brühl's, in that he believes that the site of predilection of otosclerosis is subjected to considerable mechanical strain, and that the response to this is a transformation of the normal bone into fiber bone, as in other parts of the bony system in which strains occur (cf. the Looser *Umbauzonen*). In support of this he points out the frequency of spontaneous fractures of the labyrinth in this area. He does not state that otosclerosis is due to fracture, but that the existence of spontaneous fractures indicates that there is a strain.

Wittmaack [6] wrote a vigorous attack on O. Mayer's theory. He stated that in the Looser *Umbauzonen* there is lacunar resorption, while in otosclerosis there is not. Lacunar resorption can occur in the labyrinthine bone in other conditions, such as caries and erosion from pressure of tumors, but does not occur in otosclerosis. Wittmaack does not believe in general causes for otosclerosis, such as endocrine disturbances, as he does not see why one organ is singled out. O. Mayer had attributed the disappearance of the osteocytes in otosclerosis to premature aging of the bone. Wittmaack explained this as being due to delay in the fixing fluids reaching the parts and to the powerful osmotic pressure that develops during dehydration, etc.

O. Mayer had found 60 cases in his own collection with fractures of the labyrinthine capsule which he believed were spontaneous and occurred intra vitam. Wittmaack attacked this characteristically, and stated that cracks in the specimens are not uncommon, but are more frequently found in the region of the semicircular canals than in the promontory.

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6. Wittmaack: Arch. f. Ohren-, Nasen- u. Kehlkopfh. **131**:180, 1932.

Mayer claimed to have found cells and tissue in the cracks which proved them to have occurred intra vitam. Wittmaack challenged this by stating that if they were recent they would show blood, etc., in and around the clefts. If older, they would show callus. These signs were absent. Some of the clefts had amorphous material in them, but careful focusing showed that this material was thinner than the section itself. Wittmaack attributed the presence of this material to the various solutions and osmotic pressure employed in the preparation of the specimen. He showed pictures of authentic antemortem fissures which he produced in laboratory animals several years ago. In the early stages there was always blood between the edges. After five days there was a little blood left with spindle cell fibrous tissue, the cells being arranged lengthwise to the cleft. Later still the tissue was more organized, with callus at the edge of the cleft and blood vessels crossing it.

Mayer[7] replied to Wittmaack's attack as follows: In the first place, he pointed out that Wittmaack's theory of stasis presupposes that the change occurs in old bone. This is disproved by the fact that the fibrils of the focus stop short at its edge and do not pass into the surrounding bone. This is clearly seen on examining the specimens by polarized light. This proves that the focus is new bone. Mayer further stated that Wittmaack had misinterpreted the histologic picture. He had mistaken young fiber bone for old and the old lamellar bone for young bone.

He criticized Wittmaack's experiments on fowls in that the capsule of the fowl is quite unlike that of man. It is very thin and consists of only one layer instead of three as in man. Further, it contains numerous vascular channels which pass through from side to side.

His own theory rests on three facts: (1) aging of the labyrinth capsule, shown by sclerosis, narrowing of the vascular spaces, and loss of the osteocytes; (2) spontaneous fractures; (3) the shape of the capsule and the localization of the foci at the points of greatest tension, such as the promontory and internal meatus.

There are a general or constitutional factor and an exciting factor to be taken into consideration.

Mayer was convinced that the fissures occurred ante mortem, and he showed excellent photomicrographs in which tissue with nuclei was seen. He pointed out that many authors had noted the absence of callus in fractures of the temporal bone and had speculated on the reason. It is well known that such fractures heal only by fibrous union. These fractures relieve strain and occur only at points at which otosclerotic foci are found.

A different type of paper is that of Gray,[8] who made an intensive study of the auditory nerve in cases of otosclerosis. His work shows

7. Mayer, O.: Arch. f. Ohren-, Nasen- u. Kehlkopfh. **133**:265, 1932.
8. Gray, A. A.: J. Laryng. & Otol. **47**:598, 1932.

that there are degenerative changes in the nerve in many cases of otosclerosis. Sections show that in a varying proportion the axis-cylinders have disappeared. The medullary sheath is absent in many and in those in which the axis-cylinder is still present it is not in the middle but at the side and in contact with the neurilemma. In many fibers the neurilemma is absent in spots, so that there is a fusion of adjoining nerve tubes into one. The spiral filament is also frequently absent. These changes are seen even in early cases in which there is no ankylosis. In a case of senile deafness there was considerable coalescing in the spaces of adjacent fibers. The axis-cylinders were present for the most part. This is in marked contrast to otosclerosis, in which they are generally absent.

In the early stage the medullary sheath undergoes degeneration, first with breaking down of the spiral neurokeratin filament. Next the neurilemma breaks down, forming spaces, and finally the axis-cylinder disappears.

In Gray's opinion, too much stress has been laid on the bony changes and too little study given to the other factors.

He considered the question of paracusis, and expressed the belief that the explanation is to be found in the changes in the cochlear nerve, especially in the lost insulation of the nerve fibers. The nerve impulse is weakened by the spread to adjoining fibers. On the other hand, in the presence of a loud noise there is so much stimulation that the fibers which are picking up speech gain impulses instead of losing them, and the paracusic patient hears better. Tinnitus is due to the irritation of the nerve by the surrounding fluids when the sheath is defective.

Neither the bony changes nor the nerve changes account fully for all the phenomena of otosclerosis. The state of the sympathetic nervous system has probably some important bearing on it. When an organ functions there is automatically a vasodilatation and increased blood supply. If this mechanism is defective there must be some degeneration.

In this connection, Muck,[9] who writes in and out of season on his so-called *Adrenalinsondenversuch,* or epinephrine probe test, has written something which bears on the function of the sympathetic nervous system. This test is said by its author to be a certain means of detecting any instability of the sympathetic nervous system. It consists in applying some epinephrine to the mucous membrane of the nose and waiting a few minutes until blanching occurs. The end of a probe is then drawn along the mucous membrane. Normally a pink line appears, but in people with an unstable sympathetic system a white line appears, which lasts up to fifteen minutes.

Since 1925, Muck has tried his test in all cases of otosclerosis, and has found it universally positive. He concludes that there must be a

9. Muck, O.: Ztschr. f. Hals-, Nasen- u. Ohrenh. **30**:375, 1932.

disturbance of the innervation of the vessels of the head. He believes that he can prove that one of the underlying factors of otosclerosis is a metabolic disturbance, as when he puts his patients on a purine-free diet the white line is much diminished, but comes back after they are allowed to eat meat again. This was shown in 37 women and girls, the test becoming negative when they were on a purine-free diet.

Turning now to the development of the labyrinth, Bast [10] reported on the resorption of the cartilage in the solid angle between the canals, where the process presents some peculiarities. In all other parts of the body the cartilage form of the future bone is penetrated by vascular buds accompanied by large monocytes or histiocytes which absorb the cartilage and immediately replace it by spongy bone. In this region, however, necrosis and breaking down of the cartilage occur in certain limited areas near the subarcuate fossa. Vascular buds from the subarcuate vessels then penetrate to these areas and absorb the remnants of the cartilage. The cartilage around the vascular buds stains poorly and is evidently nonactive. Normally, when cartilage is about to be replaced by bone the cells enlarge, and the matrix stains deeply. In this area the vascular buds do not initiate ossification, but the ossification spreads from the other centers with the normal changes in the cartilage. These advancing areas of ossification gradually surround and fill in the areas excavated by the vascular buds.

With regard to the growth of the semicircular canals, there is absorption of cartilage at the advancing edge and laying down of new cartilage on the opposite side. The canals reach their maximum growth at approximately half term. The superior canal is finished first, then the posterior canal and last the external canal.

Three cases in which round areas of unaltered cartilage were found in the labyrinthine capsule were reported by Altmann.[11] These patients had microtia and malformation of the external meatus. In one case the cartilage focus was in the roof of the internal auditory meatus at the junction of the enchondral and periosteal bone. In another case it was situated in the promontory in front of the facial canal. In a third case there was a similar focus in the great wing of the sphenoid in a 7 month old child with double meatal atresia.

Guggenheim [12] presented a masterly review of the whole subject of otosclerosis in two papers which should be read in their entirety. He considered that Wittmaack's theory is not proved. He pointed out that

10. Bast, T. H.: Development of Otic Capsule, Arch. Otolaryng. **16**:19 (July) 1932.

11. Altmann, F.: Arch. f. Ohren-, Nasen- u. Kehlkophf. **132**:297, 1932.

12. Guggenheim, L. K.: Ann. Otol., Rhin. & Laryng. **41**:903 (Sept.) 1932; **41**:1149 (Oct.) 1932.

the venous engorgement is not necessarily the cause of the bony changes, but may be the result of greatly increased activity of the bone. He considered that the capsule of birds and that of man are not comparable, and that there is a hereditary factor which Wittmaack left unexplained. He further dissented from Wittmaack's view that otosclerosis is a unique process, and that nothing is to be gained by a study of the general pathology of bone. Guggenheim expressed the belief that a study of other conditions of bone throws considerable light on the changes. He quoted Moritz Weber, who considers that the changes in osteodystrophia fibrosa (von Recklinghausen and Paget) are identical with those in otosclerosis and that the study is one of pathology of bone rather than of otology.

Guggenheim traced the evolution of the ear from the primitive otolithic capsule through the stage in which one semicircular canal was developed and then through to the formation of the saccule and later of the lagena. At this time there were no round or oval window and no ossicles. In late amphibian or early reptilian days the windows and ossicles made their first appearance. In other words, the cochlea, windows and ossicles developed millions of years later than the vestibular apparatus.

The mesenchymal cells which form the otic capsule are probably different from other cephalic mesenchymal cells, and are specifically destined to form the otic capsule with its windows, etc.

It is the theory of regression that the persisting mesenchymal cells retain a potentiality for differentiation and that in otosclerosis they are activated by the regressive trait of forming capsule as it is formed in lower vertebrates with no oval or round window.

Guggenheim further stated:

In certain individuals there exists a transmitted regressive trait which leads to a secondary embryonic mesenchymal activity with invasion of cartilage and then bone, and resulting in a wild proliferation of mesenchyme, a differentiation into fibroblastic tissue and finally fiber-bone. The oval window with the footplate derived from the capsule and nonexistent in certain lower vertebrates is obliterated; the footplate returns to the tissue whence it was derived.

The fissula ante fenestram, in which otosclerosis often begins, is a vestigial structure which may show cartilage throughout life and is therefore predisposed to mesenchymal invasion. The primary involvement being of a vestigial structure suggests its regressive character.

McKenzie [13] contributed an interesting article based on the analysis of 600 private cases. He divided them into three groups: a primary group in which there was no history of otitis media; a secondary group associated with otitis media and possibly induced by it, and a third group which he called incipient otosclerosis, in which there had been no suppuration or catarrh but slight deafness of long standing.

13. McKenzie, Dan: J. Laryng. & Otol. **47**:92, 1932.

The sex distribution in all three groups agreed with the general impression that females predominate.

The age of onset differed in the three groups. Thus, in the primary group 15 per cent of the cases began before the age of 15, but 50 per cent began between the ages of 15 and 35 years. The secondary group had its peak five years later than the primary group and had a secondary peak in the age period from 45 to 49. In the incipient group the peak was five years later again. The age of onset was plotted separately for males and females, and showed similar curves. Both groups had a small secondary rise between the ages of 40 and 44. There was nothing in the chart to indicate peculiar metabolism in the female as the cause.

The time in which the deafness became severe after it was first noticed averaged three and a half years. There was an enormous variation in the rate of progression.

With regard to heredity there was a positive history in 44 per cent and a negative history in 51 per cent, while in 5 per cent it was doubtful.

Tuning fork tests showed lengthened bone conduction in two thirds of the cases and shortening in one third. In the incipient cases bone conduction was shortened more often than not.

The Rinne test was always negative. The upper tone limit was lowered in 77 per cent of the primary and in 81 per cent of the secondary cases. The perception of *C* 64 was invariably diminished.

Vertigo was present in roughly one third of the cases.

McKenzie came to the conclusion that the appearance of the ear drum has no relation to the degree of hearing.

He considered catarrh at length, and stated that the group in which there is a definite history of catarrh is larger than that without catarrh. He had divided the primary from the secondary cases by the absence of manifest catarrh. However, in a number of primary cases the onset of the deafness dated from a cold or influenza. McKenzie believes that many cases begin with an inflammatory process which later disappears without a trace, and that it is quite possible that otitis media may, after all, be the original exciting factor in otosclerosis.

In McKenzie's experience otosclerosis is seldom associated with anemia, and not particularly often with rheumatism and ostearthritis. There is little evidence that the disease is due to a distant septic focus.

He believes, with most other students of the subject, that otosclerosis is brought about by two factors, a general and a local, but that even if it represents a general predisposition it requires some local cause to start it.

In 1927 Drs. Crockett and Aub, of Boston, were given a grant from the American Otological Society for the study of otosclerosis, and at the annual meeting of the society in 1932, Dr. Crockett [14] made his report.

14. Crockett: Tr. Am. Otol. Soc. **22**:47, 1932.

Twenty-three patients had been studied for four years. Their metabolism was carefully studied for one month in the hospital, where they were put on a diet especially low in calcium. The output of calcium in the excretions was measured. They were kept for a month on a diet which contained only 100 mg. per day, and in addition were treated with parathyroid extract-Collip and later with ammonium chloride. After the month the diet was relaxed somewhat, but milk, eggs, ice cream and milk compounds were avoided for four years.

Under this treatment the older cases had shown no progression of the disease, while in the control cases the patients had become decidedly worse. In the others there had been no increase in deafness. In some of the early cases a marked improvement in hearing had been obtained.

Behrendt and Berberich [15] reported some chemical studies on otosclerosis. Although it is generally believed that there is a constitutional basis for otosclerosis, up to the present the positive evidence has been slight. Various workers—for instance, Leicher and Berberich—have shown that there is a diminution of blood calcium, while Stern showed that there is a lowering of the uric acid in the blood and an alteration of the albumin-globulin fraction. Behrendt and Berberich investigated the total acid-base relationship and found that there is a lowering of the alkali reserve and of the lactic acid content. The base variation either is lowered or remains within the low normal limit. The normal level can be restored by sunlight. Behrendt and Berberich do not consider this an ordinary acidosis.

Grahe and Griebel,[16] investigating the chemical changes in otosclerosis, found that the calcium and phosphorus content of the labyrinth is distinctly higher than that of the rest of the temporal bone; further, that calcium resorption of the labyrinthine bone is specially active in otosclerosis. They came to the conclusion that absorption could be brought about only by ferments which had a specific affinity for the tissues. According to the character of the chemical tests these could only be phosphatases. Their researches seemed to show that a phosphatase specific for labyrinthine bone is active in the blood of patients with otosclerosis and produces the well known changes in the labyrinth. Their methods were of a delicate nature involving exact microchemical and analytic work.

PATHOLOGY OF THE LABYRINTHINE CAPSULE

Brunner [17] studied a case of Paget's disease of the temporal bone. He mentioned the cases described by O. Mayer, Nager, Brunner and

15. Behrendt and Berberich: J. Laryng. & Otol. **47**:502, 1932.
16. Grahe and Griebel: Internat. Zentralbl. f. Ohrenh. **35**:131, 1932.
17. Brunner, H.: Ztschr. f. Hals-, Nasen- u. Ohrenh. **30**:110, 1931.

Fischer, but was apparently unaware of the cases reported by Jenkins in 1923. This case was characterized by the extraordinary growth of fibrous marrow, which had almost entirely replaced the temporal bone. The cavity of the middle ear was not encroached on to any extent, and the mucous membrane was practically normal. The tegmen was very thick and affected with Paget's disease. The chondroid supporting tissue of the window edge was much reduced in front by the new bone, but was normal behind. The stapes and the round window were normal. There was a crack filled with fibrous tissue in the promontory leading into a marrow space. The ossicles contained large marrow spaces in which there was calcified cartilage, which is not normally present there. The internal auditory meatus was narrowed by affected bone throughout. Apart from some atrophy of the ganglion cells of the basal coil and some serous exudate, the inner ear showed nothing noteworthy.

The affected bone showed a typical structure of fibrous marrow with blood vessels here and there. Scattered through it were soft bony trabeculae composed of fiber bone. The osteocytes were well preserved, but showed few canaliculi. Numerous osteoblasts and osteoclasts were scattered around. Here and there in the mastoid portion of the bone were cysts representing the remains of mastoid cells.

In places the capsule of the inner ear was much reduced, so that there was only a thin line of blue-staining bone between the spiral ligament and the diseased bone. The resorption of the old bone appeared to be effected by osteoclasts.

Five patients with osteomalacia of the temporal bone were examined by Krainz.[18] In 3 cases the bones showed normal pneumatization, while in 2 the pneumatization was slightly reduced. In 2 cases parts of the general skeletal system were also examined. The tip of the petrous pyramid showed no special changes, but in the walls of the carotid canal there were areas of decalcified bone. In the marrow spaces in this area there was some change into fibrous marrow. The enchondral labyrinthine bone was practically unchanged throughout, and the tympanum and ossicles were almost normal. Most of the pathologic changes were seen in the periosteal bone covering the labyrinthine capsule. The floor of the middle fossa just under the dura showed considerable osteofibrotic change. The lymphoid and fatty marrow had become transformed into fibrous marrow containing incompletely formed bony trabeculae. There were many engorged veins with hemorrhages here and there. These changes were most marked in the tegmen, around the eustachian tube and in the wall of the sigmoid groove. In some cases almost the whole of the tegmen was softened. The cortex was little changed.

The pulsation of the carotid and of the dura probably accounted for the softening in the areas most affected.

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18. Krainz, W.: Ztschr. f. Hals-, Nasen- u. Ohrenh. **29**:153, 1931.

Ruttin [19] described the ears in a case of achondroplasia. The patient, aged 53, had had normal parents. He had married a normal woman and had had 8 achondroplastic children, of whom only 2 survived. He had been deaf in the left ear since childhood, but had heard fairly well in the right ear till a few years previously, when he had a severe accident with bleeding from the nose and ears. Since then he had been deaf in the right ear also. There had been a discharge from the left ear for a year before death. Death was due to peritonitis and other conditions.

The points of interest in this case are that the semicircular canals were abnormally placed, the external canal being more steeply inclined than usual and the vertical canals projecting into the cranial cavity as in infancy. This prominence of the canals was caused by the absence of the usual layer of periosteal bone covering them, the dura lying directly on the enchondral bone. The capsule bone was pierced by a striking number of new haversian canals, especially in the region of the semicircular canals. The stapes on each side was displaced inward, and there were some abnormalities of the vestibule, the crus commune being widely dilated on one side and the end of Reissner's membrane being attached to the stapes on the other. The nerve elements of the cochlea were greatly reduced or absent on both sides.

Greifenstein [20] described a case of Schüller-Christian's disease, a condition which is characterized by defects in the bones of the skull, accompanied by exophthalmos and diabetes insipidus. A woman aged 42 had had the menopause at 30, when the diabetes insipidus started. She was in the habit of drinking 10 liters of fluid every twenty-four hours. For five years she had had swelling of the right upper eyelid and frontal headaches. In the last few years a thickening had developed in the right temple and a lesser one in the left. There had been a discharge from the left ear for several years, without pain. In 1931 she had a severe dizzy spell, lasting for several weeks, and the discharge from the ear recommenced. A polyp was removed from the left ear, and the patient was found to be quite deaf in that ear. She was very stout with protruding eyes. The heart, blood, lungs and eyes were normal. A soft swelling was found on the side of the head. Roentgenograms showed large bony defects in the skull. The right ear drum was lusterless and dull. The left ear was full of granulations, was totally deaf and did not respond to the caloric test. Roentgen examination showed that the sella turcica was normal and that the bones of the face had irregular light areas.

This disease is regarded by most observers as a xanthomatosis. There is marked and constant hypercholesteremia. Fat is deposited in the reticulo-endothelial system. Granulation tissue is formed in the bone

19. Ruttin, E.: Ztschr. f. Hals-, Nasen- u. Ohrenh. **29**:135, 1931.
20. Greifenstein, A.: Arch. f. Ohren-, Nasen- u. Kehlkopf. **132**:337, 1932.

and on the dura mater, with characteristic "foam cells." These granulations obtrude into the orbit. Most of the cases occur in children and chiefly in males, and the head is the only part of the skeleton affected. This patient had 250 mg. of cholesterol per hundred cubic centimeters of blood. In 26 cases reported in the literature aural disturbances were common and various. There is evidently some connection between diseases of the ear and xanthomatosis. Xanthomatous masses are found in the ear, and they show marked eosinophilia. Foam cells full of oil droplets are common. The masses frequently break through into the external meatus and cause a foul-smelling discharge.

Hlaváček and Andres [21] reported before the Czechic Otolaryngological Society on the calcium curve after the injection of parathyroid extract in Recklinghausen's disease. Osteodystrophia fibrosa is due to an abnormal activity of the parathyroid glands, often from an adenoma. Excision of the tumor influences the disease favorably.

The authors had 2 patients whose calcium curves were plotted after 6 normal persons were tested as controls. It was found that the calcium curve was much flatter than the normal, probably owing to the fact that there was less calcium available in the bones for rapid mobilization.

TREATMENT OF OTOSCLEROSIS

Rosenfeld [22] stated that he regards otosclerosis as a local form of osteomalacia. He makes use of epinephrine, which is supposed to have a beneficial effect on osteomalacia by laying down calcium in the diseased bone and by stimulating the auditory nerve. He recommended minute doses (from 0.05 to 0.2 mg.) and claimed to have obtained improvement in the tinnitus.

Moulonguet [23] operated on the external semicircular canal in 2 cases of otosclerosis. His technic differs from that of Sourdille, in that he uses the skin of the eyelid for the skin graft. The graft is held in place for eight days with paraffin gauze.

Kopilowitsch and Zuckermann [24] have been experimenting with high frequency currents in the treatment of deafness. They gave a detailed description of an apparatus which produces from 19,000 to 50,000 vibrations per second. Exposures lasted from three to ten minutes on each side, and from two to eight sittings were given at intervals of from one to five days. Two series of patients were treated, the first including 20 patients with chronic adhesive processes, ostosclerosis, chronic suppuration of the middle ear and inner ear deafness and others in whom a rad-

21. Hlaváček and Andres: Internat. Zentralbl. f. Ohrenh. **35**:253, 1932.

22. Rosenfeld: Med. Klin. **28**:77, (Jan. 15) 1932.

23. Moulonguet: Ann. d'oto-laryng., February, 1932, p. 132.

24. Kopilowitsch, E. A., and Zuckermann, M. A.: Arch. f. Ohren-, Nasen- u. Kehlkopfh. **131**:208, 1932.

ical mastoid operation had been performed. Considerable improvement was obtained in 10 cases, or 50 per cent. The patients were tested with the whisper before and after treatment. The second series of 22 cases were of much the same type. These patients were tested with the Edelmann-Bezold continuous series of tuning forks in addition to the whisper and voice. The best results were obtained in patients with chronic suppuration of the middle ear. Of these only 2 remained unimproved. The cases of chronic adhesive catarrh also showed improvement, though not to the same extent as the suppurative cases. The cases of otosclerosis, inner ear deafness and old mastoid disease did not show improvement. There was no change in the results of the Weber, Rinne and Schwabach tests.

PARACUSIS

Jenkins,[25] in a paper on paracusis, brought up some interesting points. He stated that paracusis occurs only in patients with the otosclerotic syndrome. He drew a distinction between otosclerosis and chronic catarrh of the middle ear, in that in otosclerosis the conversational voice and whisper are heard at approximately the same distance, while in catarrh the conversational voice is heard better than the whisper. Otosclerotic patients hear well over the telephone or with electric aids, while patients with catarrh of the middle ear do not. He tested a number of patients with otosclerosis in a large quiet room, and found that there was a difference of about 30 feet (9.1 meters) between the distance at which the voice was heard and that at which it was understood. A normal person can hear a whisper at about 32 feet (9.7 meters).

With tuning forks there is often a slight loss of bone conduction for the low tones in early otosclerosis. The loss of hearing in otosclerosis is mainly in the low tones, and female voices are much better heard than male voices.

Paracusis is explained by the fact that the disturbing sounds are very low pitched. The patient with otosclerosis does not hear them, but the normal person does. Furthermore, in a noise a normal person not only increases the volume of the voice, but also raises the pitch, which brings it into a tone scale in which the otosclerotic patient would hear it better in any case. Thus, paracusis is not really paradoxical.

MIDDLE EAR DEAFNESS

Mygind [26] expressed the belief that chronic catarrh and an adhesive process are related, and that the underlying condition is edema of the mucous membrane producing a condition similar to that in the fetus. The edema is intracellular and is part of a general retention of water in

25. Jenkins, G. J.: J. Laryng. & Otol. **47**:517, 1932.
26. Mygind, S. H.: Acta oto-laryng. **16**:333, 1931.

the tissues. The condition is best treated in the early stages by dehydration by means of diuretics (mersalyl) and restriction of the intake of fluids.

TINNITUS

Bárány [27] classifies ear noises as (1) subjective, (2) objective and (3) subjective-objective. In the last type there is an actual noise which is too faint for the observer to hear. An example is that of a woman on whom Bárány was operating under local anesthesia for obstinate vertigo and pulsating tinnitus. As he was trimming the facial spur the artery there was wounded and had to be stopped with paste. The patient immediately informed him that the tinnitus had stopped. Later the vessel was again wounded, and the tinnitus immediately recommenced, but stopped again when the bleeding was arrested.

According to Jacod,[28] bacteremia from Bacillus coli produces not only local metastases but also vasomotor disturbances. He had noticed for a long time the influence of intestinal disturbances on the noises in the ear and the slight vertigo of which certain patients complained. He cured 2 patients with tinnitus by the administration of colon bacillus vaccine for gastro-intestinal disturbances. He now always has the feces examined for excessive numbers of B. coli, and treats the patients with vaccine. He believes that the toxin produces a congestion of the inner ear.

Babbitt [29] reviewed the numerous known causes of tinnitus, and came to the conclusion that there can be no routine treatment for a condition the etiology of which is so varied.

An interesting and unusual case is presented of a young man on whom many forms of treatment had been tried without avail. Thorough local and general examination revealed nothing to account for the tinnitus. Some temporary relief was obtained by incising the drum along the edge. Later the patient accidentally discovered that by putting a finger in the ear and withdrawing it, producing suction, the tinnitus was temporarily relieved.

The author obtained complete relief for the patient by turning down a flap from the ear drum, dividing the stapedius tendon, breaking down adhesions in the attic and cutting the tensor tympani. The handle of the malleus was then pulled outward a little.

PATHOLOGY OF THE INNER EAR

Finding very little in the literature on the pathologic changes in arteriosclerosis of the internal auditory artery, Wangemann [30] made a

27. Bárány, R.: Acta oto-laryng. **16**:311, 1931.
28. Jacod, M.: Ann. d'oto-laryng., February, 1932, p. 134.
29. Babbitt, J. A.: Ann. Otol., Rhin. & Laryng. **16**:21, 1932.
30. Wangemann, H.: Ztschr. f. Hals-, Nasen- u. Ohrenh. **30**:135, 1932.

study of 100 internal auditory arteries. The material was obtained from 50 unselected autopsies. The vessels were studied in situ and their appearance compared with the state of the general vascular system, the coronary vessels and the basal vessels of the brain. The study was limited to the free course of the artery, i. e., between the basilar artery and the internal auditory meatus. In 100 arteries arteriosclerotic changes were found 29 times. Cases were included in which the only change was tortuosity of the vessel. In 13 instances the sclerosis was bilateral. In no case were the changes marked. There is certainly no predilection for arteriosclerosis in the auditory arteries as compared with other vessels. As a contrast to the involvement of the auditory arteries in 29 per cent there was involvement of the basal vessels of the brain in 88 per cent, of the coronary arteries in 88 per cent and of the aorta in 90 per cent in the same material.

Fineberg,[31] discussing cholesterol metabolism in general, pointed out that patients with hypertension, arteriosclerosis, apoplexy, chronic nephritis and nerve deafness all have definite hypercholesteremia. The cholesterol in the blood is normally from 140 to 180 mg. per hundred cubic centimeters. An increase in blood cholesterol may be due to excessive ingestion, to cell destruction or to endocrine disturbances. Fineberg and Berberich had shown in a previous article that inner ear deafness is the rule in apoplexy and hypertension. They had examined a series of 19 patients histologically. Sixteen had hearing tests before death, and all showed inner ear deafness. All showed an arcus lipoides myringis. Lipoid globules were demonstrated in the drum membrane and around the eustachian tube. They were found only in the middle ear and mastoid in cases of inflammation. Droplets were also seen in the stria vascularis and in the organ of Corti.

Some feeding experiments were carried out on rabbits with large amounts of cholesterol. This produced an increased blood content, and the blood pressure was raised. Examination of the ears showed greatly increased lipoid in the cartilage of the ear, the ear drum and all parts of the cochlea. The conclusion was reached that cholesterol metabolism plays a great part in certain types of inner ear deafness.

Following up the work of Fineberg, Thaler [32] picked out 30 patients in the medical ward with various conditions, but all having increased cholesterol in the blood, to see if there was any loss of hearing. The ages ranged from 17 to 61 years. There was a decided loss of hearing in this group as compared with the average normal values compiled by Bunch. Thaler concluded that systemic disease by itself does not produce deafness but that in conjunction with a raised cholesterol content of the blood it produces a progressive loss, increasing with age.

31. Fineberg, M.: Laryngoscope **42**:177, 1932.
32. Thaler, N.: Laryngoscope **42**:741, 1932.

Fraser [33] divided cases of deaf-mutism into two broad groups: (1) those due to an error of development and (2) those due to inflammation or trauma. The first group can be subdivided into sporadic and endemic forms and the second group into forms caused by trauma, congenital syphilis, meningitis and labyrinthitis, and labyrinthitis caused by otitis media.

Sporadic congenital deafness may be due to: 1. Total aplasia of the labyrinth (Michel type); this is very rare. 2. Malformation of the bony and membranous labyrinth (Mondini or Alexander type). The bony capsule of the cochlea is flattened, and only the basal coil is present. The apex is a wide space. 3. Malformations affecting both cochlea and vestibule. 4. Sacculocochlear malformations (Scheibe). In this form the utricle and canals are normal. The organ of Corti is absent here and there, the membrana tectoria is out of place, and the spiral ganglion is undeveloped. This is the most common type, comprising 70 per cent of all cases of congenital deaf-mutism.

Endemic deafness of the cretinous type is common in Switzerland. Fraser quoted Nager as stating that 29 per cent of cretins are deaf-mutes, and 32 per cent are hard of hearing. This type shows myxomatous swelling of the middle ear, filling up the niches, along with frequent malformations of the ossicles. The vestibule and canals are usually normal, but the organ of Corti is degenerated.

Of the cases of acquired deaf-mutism, about 3 per cent are due to trauma from old fracture. Hemorrhages into the scalae are replaced by fibrous tissue and new bone. Congenital syphilis accounts for a certain number, either from an otitis media spreading through to the labyrinth or from a meningitis with secondary labyrinthitis. Cerebrospinal meningitis, measles and mumps cause a certain number. In cases of old labyrinthitis from otitis media one frequently finds closure of the round window by new bone. The vestibule shows dilatation of the utricle and saccule and connective tissue in the perilymphatic space.

Turner and Fraser [34] devoted a third of their series of papers on labyrinthitis to the results of treatment. Their investigation is based on patients treated in the Royal Infirmary, Edinburgh, in the years from 1907 to 1932. The cases with labyrinthine complications numbered 216, or 1.5 per cent of all cases of suppuration of the middle ear. This included 22 cases of postoperative or "induced" labyrinthitis. The number of spontaneously occurring cases was 194, or 1.4 per cent. Labyrinthitis occurred in 0.6 per cent of the acute cases and in 1.8 per cent of 10,381 chronic cases.

Intracranial complications were already present in 25 per cent on admission to the hospital. These included 25 cases of leptomeningitis,

33. Fraser, J. S.: J. Laryng. & Otol. **47**:538, 1932.
34. Turner, A. L., and Fraser, J. S.: J. Laryng. & Otol. **47**:657, 1932.

15 of transverse sinus thrombosis, 3 of cerebellar abscess and 6 of abscess of the temporosphenoidal lobe. In this connection the authors stated that they have never held the view that there is a close association between labyrinthitis and cerebellar abscess.

Of the patients with spontaneous labyrinthitis not associated with intracranial complications on admission, 97.3 per cent recovered, while of the group with induced labyrinthitis 63.6 per cent recovered. Of the 50 patients with intracranial complications, 26 per cent recovered. With regard to secondary or induced labyrinthitis there is sometimes a difficulty in distinguishing between a serous and a purulent labyrinthitis. Turner and Fraser, as a result of their experience, believe that it is much wiser to take the patient at once to the operating room and perform a Hinsberg operation than to wait a day or two. Those operated on promptly usually recovered while those with whom there was some delay succumbed.

Hagens [35] described in detail the ear of a child dying forty hours after birth and showing an absence of the external auditory meatus and auricle. A bony plate continuous with the squama occupied the situation of the drum membrane. The internal auditory meatus was represented by a dimple filled with connective tissue. Microscopically examined, the malleus and incus were present behind the plate of bone, and the cavity of the middle ear was filled with embryonic tissue. The most interesting feature of the case was the presence of a mass of thymus tissue dividing the middle ear into two cavities. This mass showed a suggestion of cortex and medulla and several Hassall's corpuscles. A large vein was found running along the inner wall of the middle ear and emerging in the mastoid region. (This is probably a persisting remnant of the large vein seen in this situation in 8 week embryos.—*Abstractor.*) The vestibule and cochlea were well developed, though no nerve fibers seemed to reach the cochlea. In the brain the motor nucleus of the facial nerve was absent, though the sensory part was well developed.

Referring to a controversy between Demetriades and Schlittler as to whether there occurs a homolateral degeneration of the labyrinth in cases of carcinoma, Brunner [36] reported a case of this kind. In a woman dying of carcinoma severe deafness, loss of some of the reflexes, diplopia and absence of the caloric reaction developed before death. Microscopic examination of the left ear showed no tumor in the internal meatus. Nothing was noted in the middle ear, but there were severe atrophy of the spiral nerve and ganglion in the basal coil of the cochlea, slight atrophy in the middle coil and degeneration of the apical coil.

35. Hagens, E. W.: Malformation of Auditory Apparatus in New-Born, Associated with Ectopic Thymus, Arch. Otolaryng. **15**:671 (May) 1932.

36. Brunner, H.: Ztschr. f. Hals-, Nasen- u. Ohrenh. **30**:443, 1932.

There was some atrophy of the nerves of the cristae, but no evidence of labyrinthitis. Brunner attributed the nerve degeneration to the severe degree of cachexia.

Dederding has been studying Ménière's syndrome for some time, and has given her views in two papers.[37] The treatment is founded on the theory that these patients suffer from a general disturbance of fluid balance, which shows itself in various ways, such as headaches, cold feet and hands, congestion, rheumatism, gastro-intestinal disturbances, tiredness, irritability, depression and sleeplessness. This is brought about by vasomotor disturbances and failing capillary function with excessive transudation. Rapid changes in fluid tension cause edema in the subcutaneous tissues and intermittent changes in pressure in the labyrinth.

Many of the patients feel much better when they are exercising in dry weather and sweating, and are not so well during sedentary periods, especially during mealtime when considerable fluid is ingested.

Therapeutic measures consist in a salt-poor diet and restricted intake of fluids (700 Gm. a day). The food must not be poor in calories. Baths, massage, gymnastics and administration of diuretics, especially mersalyl, all help the necessary dehydration. The patients are also treated locally *per tubam*.

The results of this treatment have been good. Of 157 patients with Ménière's syndrome, only 7 had nystagmus and giddiness on discharge. Of 111 patients with reduced hearing, 85 showed improvement. In a check-up of 83 patients two years later, only 16 were found to have had attacks of giddiness. Most of the recurrences were due to breaking the regimen.

Thacker Neville[38] is a supporter of the theory of Dederding that vertigo, etc., may be due to faulty water metabolism. He mentioned thickening of the skin of the back of the neck and calves, headache and rheumatism as being brought on by this general disturbance. The cause is said to be overloading of the tissues with water. The variability of the deafness and vertigo may be due to the variable amount of fluid in the tissues.

To test the metabolism, the patient is given 1,000 cc. of water to drink, and the output is measured for four hours. A normal person will pass 1,000 cc. or more, while one of these patients may pass only 500 or 600 cc., with a corresponding increase in body weight. The second test consists of administering 1 cc. of mersalyl intravenously. It produces increased diuresis on the following day. A normal person has a diminished output on the third day, but a waterlogged person will show an increased diuresis. The treatment is lifelong and consists in a salt-

37. Dederding, D.: Ugesk. f. læger **93**:497, 1931; Acta oto-laryng. **16**:404, 1931.

38. Thacker, Neville, W. S.: J. Laryng. & Otol. **47**:533, 1932.

free diet, restricted intake of fluids and increased elimination. Bulbocapnine is recommended if the patient should take too much water.

Vogel [39] defined Ménière's syndrome as a noninflammatory labyrinthine disease with a negative Wassermann reaction and negative neurologic findings. This definition includes cases of dizziness with objective findings in the cochlea and vestibule, such as deafness, spontaneous nystagmus, head nystagmus and gross changes in the excitability of the vestibular organs. All cases of injury are excluded.

One hundred patients were studied, of whom 51 were men and 49 women. Of these 5 suffered from hay fever and 9 from urticaria after taking various foods. No association could be traced between the attacks of Ménière's syndrome and the urticaria or hay fever. Thirteen patients had nonallergic vasomotor rhinitis, 6 asthma, 20 cholecystitis, 6 gastric or duodenal ulcer, 14 migraine and 6 epilepsy. In all, 47 had some condition of this sort. The number of cases of disease of the gallbladder is striking. In several instances the patients had suffered for years from migraine, which was replaced by attacks of Ménière's syndrome later in life. Four men and 10 women had a combination of migraine and attacks of Ménière's syndrome. Vogel concluded that Ménière's syndrome is not an allergic manifestation but that it is associated with a stimulation of the vegetative nervous system, which seems to predispose the patient to these attacks.

Fremel [40] reported the case of a man with symptoms of Ménière's syndrome due to a tumor of the medulla. A man aged 32 had had dizzy attacks for thirty months at weekly intervals. He had no disease of the inner ear. He was seen in an attack when vertical nystagmus occurred. In the next few weeks signs of a tumor of the medulla developed. He never had any disturbance in hearing.

Postmortem examination revealed a glioma extending from the second cervical segment to the abducens nucleus. The vestibular nucleus itself was not affected, though the tumor was very near it.

TRAUMA OF THE LABYRINTH

Wittmaack,[41] in a long article, described the effects of trauma on the inner ear. The generally accepted view is that following trauma there occur hemorrhages into the inner ear which are later replaced by fibrous tissue, with atrophy of the end-organs and nerve fibers. Wittmaack has a new explanation for the degeneration of the inner ear. According to his theory, when a sudden shock is transmitted through the oval window to the inner ear the delicate structures of the organ

39. Vogel, K.: Ztschr. f. Hals-, Nasen- u. Ohrenh. **29**:175, 1931.
40. Fremel, F.: Ztschr. f. Hals-, Nasen- u. Ohrenh. **29**:207, 1931.
41. Wittmaack, K.: Arch. f. Ohren-, Nasen- u. Kehlkopfh. **131**:59, 1932.

of Corti are caught between two columns of fluid and crushed out of existence as though between two millstones.

He described the case of a woman of 30 who at the age of 14 was hit behind the left ear by a hard snowball and thereafter was deaf in that ear. There was no bleeding from the ear or loss of consciousness, but deafness, tinnitus and occasional vertigo were noticed after that time. Histologic examination of the ear sixteen years later showed a normal middle ear and no sign of fracture of the labyrinth but complete disappearance of the organ of Corti in all the coils. There was no sign of any old inflammatory change. The end-organ of the saccule had disappeared, but the utricle and semicircular canals were normal.

The organ of Corti contains a canal under the supporting cells and another under the membrana tectoria. These two bodies of fluid do not communicate freely with the rest of the endolymph. The result is that when there is any sudden increase of pressure in the endolymph as a whole, the soft barrier of the organ of Corti is crushed out of shape. This hypothesis is intimately associated with Wittmaack's *Tonuslehre*. According to this theory the membrana tectoria is in intimate contact with the hair cells. Fluid is secreted by the cells of Corti and is held in suspension in the jelly-like membrana tectoria, the difference in fluid tension between the free and suspended fluid constituting the tonus. Wittmaack stated:

> All the protoplasmic or cellular elements of these structures, that is, not only the Corti membrane and Corti's organ but also the sensory organ in the sacculus or utriculus, must be squashed through the hydrostatic pressure effect of the endolymph on the one hand and the liquor enclosed in themselves on the other hand, as though between two millstones; that is, with sufficient pressure they become ground into a protoplasmic pulp.

Wittmaack performed some experiments on rabbits to prove his point. He exposed the oval window by operation. Then by means of a cannula with a rubber stopper around it he pushed the stapes into the labyrinth and closed the window with the cannula. By applying pressure with a syringe he gave the necessary shock to the labyrinth. With relatively slight pressure, destruction of part of the organ of Corti was produced. With greater pressure, hemorrhages and total destruction of Corti's organ with fracture of the spiral lamina resulted. The destruction of the organ of Corti was immediate. In two weeks the cochlear ganglion was completely atrophied.

Wittmaack stressed the point that this result should not be confused with the degeneration due to fibrosis, as the one occurs immediately and the other late.

Control experiments were performed in which the stapes was pushed in, but no pressure was exerted. Hemorrhages were found, but no destruction of the organ of Corti.

In reading this article the thought arises that such relatively gross and brutal injuries to the labyrinth as were done in these experiments are not strictly comparable to the injury described in the clinical case in which the deafness was due to a blow of only medium severity on the head. However, Wittmaack's theory may possibly explain the deafness due to injuries of the ear produced by explosions and similar accidents.

In a second paper Wittmaack [42] followed up the subject of injury to the sensory cells in the inner ear. He produced the same destruction of the organ of Corti by a momentary loud sound from a *C* 4 whistle transmitted through the air. There was total destruction of the organ of Corti in the basal coil with a lessening degree of damage toward the apex. The cochlear nerves and the spiral ganglion cells were destroyed immediately.

Lesions of the cochlea by bone conduction produce destruction of the sensory cells, but leave the supporting cells intact. Wittmaack fixed a guinea-pig to a turntable so that the axis of the cochlea was parallel to the radius of the table. He then turned the animal at the rate of 120 revolutions per second. This caused strong upward pressure of the basilar membrane on the organ of Corti and produced the same lesions as those caused by bone-conducted noise. He asserted that in bone conduction the impulse on the organ of Corti is in the reverse direction from that in air conduction. The impulse starts at the modiolus and is transmitted to the basilar membrane, which has to move the fluid above and below it. This absorbs much of the energy and weakens the impulse.

Langenbeck [43] distinguishes between air conduction and body conduction and considers craniotympanic conduction of no practical importance. A point that has been lost sight of in the squabbles between Wittmaack, Siebenmann and Barth is that vibrations from the floor give off air waves. The important question is whether these vibrations are heard better by air or through the body. Vibrations from the floor are largely dispersed and lost by the time they reach the head, but the air waves that they give off can be heard. Vibrations from the floor can be imitated by placing the ear piece of an oto-audion to the sole of the foot. No air waves are heard. Similarly, vibrations of the floor producing air waves can be imitated with a cardboard membrane attached to the earpiece. All the frequencies can be so tested and the intensity checked. The waves through the body were poorly heard, and even then it was difficult to distinguish them from air-conducted sounds. Under actual working conditions air-conducted sounds would be much more intense than those from 0.25 meter of cardboard. It is clear that damage by bone-conducted sound can never come into practi-

42. Wittmaack, K.: Arch. f. Ohren-, Nasen- u. Kehlkopfh. **133**:181, 1932.
43. Langenbeck, B.: Ztschr. f. Hals-, Nasen- u. Ohrenh. **30**:609, 1932.

cal consideration. Under all actual conditions the accompanying air-conducted sounds would damage the ear first, as their intensity is more than ten times, and probably one hundred times, as strong.

The author discussed the question of pathologic conditions of ears with a negative Rinne test in which air conduction is perhaps only one one-hundredth of the normal amount, while bone conduction is normal. These patients never actually hear better than normal by bone conduction; they only apparently hear better in the lower frequencies. With these persons the waves through the body are so weakened by the time they reach the ear that they are harmless.

ACOUSTIC TUMORS

De Kleijn and Gray [44] reported a case of bilateral acoustic tumor in a man suffering from multiple neurofibromatosis (von Recklinghausen). On the left side the tumor had grown into the cochlea and out through the round window into the middle ear. In this same cochlea the apex was filled by new bone, which was apparently not derived from the endosteum, as there was a distinct line of demarcation. There was no sign of inflammatory activity. In the right ear the stria vascularis showed considerable vacuolation along its whole extent. The spiral ganglion cells had completely disappeared, leaving only empty spaces. The organ of Corti was absent in the basal coil, but more or less normal in the others.

44. de Kleijn, A., and Gray, A. A.: J. Laryng. & Otol. **47**:589, 1932.

Abstracts from Current Literature

Ear

Serious Disturbances Produced by Latent Otitis and Mastoiditis in the Infant: Diagnosis and Early Treatment. I. Oreviceanu, I. Herovici and W. Grunberg, Bull. Soc. de pédiat. de Paris **30**:425 (April 2) 1932.

There are two forms of latent otitis and mastoiditis in infants: (1) acute, in which diarrhea, vomiting, dehydration and toxemia are present, and (2) subacute, in which the development is slower, the infant shows progressive loss of weight, and digestive, pulmonary or other symptoms predominate. In all of these cases the ear acts as the primary focus of infection, from which bacteria get into the blood stream; the bacteria may lodge in another part of the body, producing the complications. Early paracentesis is of the utmost importance in the treatment and if repeated paracenteses are not followed by decided improvement, mastoid antrotomy should be performed. There was a characteristic differential blood count in the infants with latent otitis and mastoiditis, which consisted of a marked decrease in the monocytes and an increase in the number of young neutrophils, with some promyelocytes.

Benjamin, Montreal, Canada. [Am. J. Dis. Child.]

Latent Otitis Caused by Hoffmann's Bacillus in an Infant. A. Rusesco and E. Daraban, Bull. Soc. de pédiat. de Paris **30**:431 (May 14) 1932.

Diphtheroid bacilli are usually considered as saprophytic organisms. In this case, that of an infant, 8 months old, diphtheroid bacilli were present in smears of the pus obtained by paracentesis of the membrane of the drum, and cultures of the pus produced a pure growth of the same bacilli. Drainage from the ear continued for about fifteen days after the incision was made.

Benjamin, Montreal, Canada. [Am. J. Dis. Child.]

Diagnosis of Otogenous Meningitis. G. Alexander, Wien. klin. Wchnschr. **45**:788 (June 17) 1932.

A careful and objective observation is necessary for the early diagnosis of meningitis. It may be mistaken for otitis even by an experienced physician. One must regard with suspicion every case of suppurative inflammation of the middle ear in which the local cause is insufficient to account for such symptoms as continued fever, headache, insomnia, unrest and dyspnea. Spinal puncture is important for the differential diagnosis and therapy, especially in the early stages. If chronic disease of the brain (neoplasm, gumma, tubercle or encephalitis) is present, only a small amount of liquid should be taken when spinal puncture is performed, otherwise severe cerebral symptoms may occur.

In meningitis the protein content of the fluid is increased; in tuberculous meningitis it is either decreased or negative, and in serous meningitis and in neoplasm it is increased.

In otogenous meningitis the spinal fluid is normal, and in abscess of the brain it is usually turbid and sterile.

Abt, Chicago. [Am. J. Dis. Child.]

Influenzal Otitis. E. V. György, Wien. klin. Wchnschr. **45**:818 (June 25) 1932.

E. V. György records four illustrative cases in infants aged from 2 to 13 months, and distinguishes the following four forms of influenzal otitis: (1) the

hyperacute toxic form, characterized by a sudden severe onset with rise of temperature to 104 F., vomiting, diarrhea, rapid development of suppurative otitis and subperiosteal abscess, followed by death in a few days in spite of paracentesis and antrotomy; (2) an acute form in which the course is not so rapid and treatment is usually successful; (3) a subacute form; (4) a protracted form. In the acute cases the aural symptoms dominate the clinical picture, and are accompanied by catarrhal involvement of the nasopharynx and lungs. In the subacute cases the catarrhal symptoms predominate, and the otitis is less prominent.

ABT, Chicago. [AM. J. DIS. CHILD.]

EFFECT OF FATIGUE OF ACOUSTIC NERVE ON HEARING TESTS. R. BLEYL, Ztschr. f. Hals-, Nasen- u. Ohrenh. **32**:62 (Sept. 16) 1932.

Bleyl stresses the importance of eliminating the factor of fatigue of the acoustic nerve in accurate determinations of the hearing capacity, and discusses the investigations that have been conducted on this point. He thinks that a more exact determination of the duration of hearing which produces fatigue and the duration of the fatigue can be attained only if a given tone is allowed to act on the ear for the desired length of time with the intensity necessary for perception (threshold value). He thinks that this might be possible with the oto-audion. He considers that fatigue of the accoustic nerve is a demonstrable occurrence, the degree of which depends on general bodily and mental fatigue. It therefore appears with particular intensity in conditions of somatic or psychic exhaustion, especially in neurasthenic or hysterical persons. It is scarcely influenced by disease of the middle ear, but is increased slightly by labyrinthine disease.

EDITOR'S ABSTRACT.

DEEPLY LOCATED INFLAMMATORY FOCI IN THE TEMPORAL BONE. W. LANGE, Ztschr. f. Hals-, Nasen- u. Ohrenh. **32**:99 (Sept. 16) 1932.

Lange discusses the symptomatology and anatomicopathology of the deep-seated inflammations of the temporal bone occurring after acute otitis media, without involvement of the labyrinth. He studied twenty cases clinically and anatomically, and observed clinically six cases in which recovery occurred. Paralysis of the abducens is not a constant sign of deep-seated inflammation in the opinion of the author. Not every inflammation of this kind leads to paralysis of the abducens; specific conditions must be fulfilled. Either the inflammation must penetrate far into the bone and the dura, or an epidural inflammation which does not quite reach the abducens must lead to subdural circumscribed inflammatory foci which reach the abducens. In the author's experience paralysis of the abducens could not be ascertained in any of the fatal cases, but was present in four of the patients who recovered. The difficulty in exposing the inflammatory focus by following the mastoid cells as they are shown in the roentgenogram lies in the obstruction of the path by the labyrinth. A normal labyrinth should not be sacrificed unless there is a pronounced meningitis, and unless there is no indication that the origin is anywhere except medial to the labyrinth. In the absence of a generalized meningitis and in the presence of a normally functioning labyrinth, one should try to find a path leading to the infectious focus. All the cells of the mastoid can never be reached, but in mastoid processes with extensive pneumatization, one can hope to create more favorable healing conditions for unapproachable cells by early than by late intervention. Only stereoroentgenograms taken from several directions can determine the degree and type of pneumatization of the mastoid.

EDITOR'S ABSTRACT.

INFLAMMATORY DISEASES OF THE MIDDLE EAR AND MASTOID CELLS. L. SINGER, Ztschr. f. Hals-, Nasen- u. Ohrenh. **32**:110 (Sept. 16); 130 (Nov. 12) 1932.

Singer presents a morphologic study of inflammatory diseases of the middle ear and the mastoid cells, including a study of the fetal and postnatal development

of the middle ear and the mastoid process with a consideration of the complete and incomplete pneumatization of the temporal bone. The study is based on the examination of seventy temporal bones and a thorough review of the literature.

EDITOR'S ABSTRACT.

ABSCESSES OF THE BRAIN. W. KLESTADT, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia oto-laryng.) **23**:94 (Sept.) 1932.

Klestadt reports three cases of abscess of the brain in which necropsies revealed conditions that were unexpected on the basis of the clinical symptoms. The first case concerned a boy with otogenous sinus thrombosis, an abscess of the temporal lobe and another in the cerebellum. About five weeks after closure of the wounds of the thrombophlebitis and the cerebellar abscess, when the fistula, which had persisted in the abscess of the temporal lobe, was finally healing, a temporal lobe syndrome appeared on the right side. The fistula was reexamined, but yielded no pus. Meningitis set in. Search for an abscess and attempted drainage of the inferior cornu of the lateral ventricle and the posterior cornu were in vain. The case terminated fatally. At necropsy, a scar 3 cm. in length was found in the cerebellum. No suppuration was found in the brain except that due to the last interventions. The author thinks that the new focal symptoms were probably due to the effects of cicatrization, and that the boy would have survived had it not been for exacerbation of the meningitis caused by the search for the abscess. The second case concerned a man with severe aphasia following acute otitis media, who died of meningitis. At necropsy, the author expected to find a voluminous abscess, because the aphasia had included practically all varieties, but instead the original abscess was only of walnut size. Since the marked regression of aphasic symptoms which followed cerebral decompression and drainage of the abscess had been followed by a recurrence of these symptoms, the author expected a severe retention of pus or a focally exacerbated encephalitis, but he found, instead, only a small, cherry-sized abscess near the major abscess, but independent of it. The third case concerned a girl with an otogenous meningitis. Radical operation exposed an extradural abscess extending from the upper bend of the sinus toward the median cranial fossa. At necropsy, a large, oval, subdural abscess was found ending about 1 cm. behind the trepanation opening and not connected with the region of the extradural abscess. It extended from the temporal lobe backward over a considerable part of the occipital lobe. The lack of focal symptoms and the general course of disease justified the conservative intervention.

EDITOR'S ABSTRACT.

Pharynx

LESIONS OF THE PHARYNX IN ACUTE RHEUMATISM. A. D. FRASER, Arch. Dis. Childhood **7**:181 (Aug.) 1932.

This report deals with the histologic examination of the pharyngeal tissues and cervical lymph nodes of four patients who died of rheumatic fever uncomplicated by any other disease. Two patients (a man, aged 20, and a boy, aged 8 years) had had recurrent attacks of rheumatic fever. The other two cases (those of boys, aged 8 and 7 years) proved fatal during the first attack.

Rheumatic nodules were found in the pharyngeal tissues. These nodules were more commonly found in the vicinity of the tonsils, radiating from the tonsillar capsule into the connective tissue of the muscles of the pharynx.

Similar lesions were present in the region of the lingual and laryngeal tonsils in one of the recurrent cases. In this case also there were significant changes in the regional lymph nodes. These changes consisted of marked endothelial hyperplasia with much polymorphonuclear and plasma cell infiltration.

Aschoff nodules were found in the connective tissue surrounding the vessels in the capsule and fibrous stroma of some of the nodes in the upper, deep cervical group on both sides.

KELLY, Milwaukee. [AM. J. DIS. CHILD.]

THE CONSERVATIVE TREATMENT OF TONSILLITIS BY THE SUCTION METHOD OF ROEDER. M. JÄGERMANN, München. med. Wchnschr. **79**:1751 (Oct. 28) 1932.

Tonsils should not be removed as frequently as they are at present because they have a protective function, and, moreover, the tendency for the development of colds continues after tonsillectomy. A simple conservative method for the treatment of acute and chronic tonsillitis is by the suction technic of Roeder. A glass suction cup at the end of a glass tube fits over the tonsil. The end of the glass tube is attached to a rubber bulb. By proper manipulation infected material can be sucked out of the tonsillar crypts. The frequency of the treatment depends on the type and duration of the disease and the condition of the tonsils. Usually, suction is performed two or three times a week for several weeks.

BRAHDY, Mount Vernon, N. Y. [AM. J. DIS. CHILD.]

ROENTGEN TREATMENT FOR DISEASES OF THE TONSILS. G. SCHULTE, Strahlentherapie **36**:708, 1930.

The author has been irradiating tonsils since 1924. He tried to find the smallest effective dose in order to avoid injuring the many lymph glands of the region which are particularly sensitive to irradiation. The patient lies on his back with his head turned to one side. The tube is placed at the angle of the jaw and the rays directed upward and backward in order to strike the pharyngeal as well as the palatal tonsil. The focus skin distance is 30 cm., the field being 6 by 8 cm.; children are given 140 kilovolts, filtered through 4 mm. of aluminum (⅓ skin erythema dose [147 roentgens]); adults, 210 kilovolts, filtered through 1 mm. copper and 1 mm. aluminum (⅓ skin erythema dose [170 roentgens]). The second irradiation from the other side is given in the same way the next day or the day after in order to avoid too intense reactive swelling. Homogeneous irradiation of the whole pharyngeal ring is not used. The same cycle of irradiation is repeated after four weeks and again in eight weeks if necessary. In the course of six years the author has irradiated 257 patients, 132 children and 125 adults; 90 per cent of these were cured, 6 per cent were improved and 4 per cent were not improved. Large, hard fibrous tonsils did not contract much; the author now generally operates on such patients. Softer hypertrophied tonsils return to their normal size. The treatment is particularly effective in chronic recurrent anginas. Several patients who had chronic recurrent polyarthritis, the attacks generally following attacks of angina, were relieved. Roentgen treatment has an advantage over tonsillectomy in that it does not require anesthesia and there is no shock, loss of work, danger of primary or secondary hemorrhage, possibility of infection or pulmonary complications or aural infections. It is often impossible to perform operation, while roentgen treatment is always possible.

MORGAN [AM. J. ROENTGENOL.]

REFLEX TEMPERATURE CHANGES OF NASAL AND PHARYNGEAL MUCOSA FROM LOCAL APPLICATION OF COLD. W. UNDRITZ and R. SASSOSSOW, Ztschr. f. Hals-, Nasen- u. Ohrenh. **32**:300 (Dec. 22) 1932.

Undritz and Sassossow state that the application of cold to the abdominal skin of dogs and guinea-pigs causes a decrease in the temperature of the nasal and pharyngeal mucosa and especially of the tonsils. This reflex drop in temperature occurs earlier than the lowering of the body temperature and is greater, varying between 2 and 4 degrees. The vascular contraction which can be produced by the sympathetic nervous system in distant parts results in a cooling of the mucosa where it is exposed to the circulating air; that is why the phenomenon can be best observed on the surface of the tonsillar and oral mucosa (with open mouth) or in the front part of the nasal cavity. In the trachea, nasopharynx and similar structures, the drop in temperature is less. The inhaling of cold air is sufficient to lower the temperature of the tonsillar mucosa. The reflex vascular contraction

of the arteries is not the only factor in the production of the decrease in temperature; a retarding of the circulation and, possibly, an interference with local production of warmth by trophic reflexes are also factors. If sufficiently intense, the cooling of the mucosa causes a decrease in the resistance of the tissue which, particularly in the tonsil, which always harbors pathogenic bacteria, may lead to inflammation. This explains why the occurrence of tonsillitis following nasal operations increases in periods of cold and changing weather. The oral respiration necessitated by the nasal tamponade also plays a rôle. Oral respiration favors the occurrence of tonsillitis.

EDITOR'S ABSTRACT.

EXPERIMENTAL HISTOLOGIC STUDIES OF THE TONSIL. R. WALDAPFEL, Ztschr. f. Hals-, Nasen- u. Ohrenh. **32**:400 (Feb. 12) 1932.

On the basis of an extensive experimental investigation on human beings and on animals, Waldapfel states that substances introduced into the mucosa of the inferior nasal concha reappear in the tonsils, where they can be demonstrated microscopically and to some extent chemically; substances introduced into other parts of the nasal mucosa are conveyed exclusively to the regional lymph nodes. Organic material reaches the tonsils in larger amounts than inorganic material, cellular material in still larger amounts and bacteria most abundantly. The coloring matter is distributed in varying amounts in different parts of the tonsil. The substances are conveyed from the nose into the tonsil by the efferent veins of the cavernous plexus of the inferior conchae which communicate with the veins of the pharynx, which in turn communicate with the tonsils. As the veins of both sides of the pharynx communicate with each other, the substances injected unilaterally into the concha appear in both tonsils. Following intravenous injections in other parts of the body, a small part of the injected material reaches the tonsil via the blood stream, but it is a much smaller proportion than that carried by the venous route from the cavernous plexuses of the nose. The substances which reach the tonsils are eliminated into the crypts through the epithelium, usually where it is reticulated but also through intact portions. From six to twelve hours after intravenous injection the injected substance can be demonstrated in the crypts. Only small particles of bacterial size are eliminated. A part of the material entering the tonsil is conveyed to the regional lymph nodes. These experimental results confirm the opinion of those who attribute to the tonsillar tissue the function of ridding the blood stream of some of the foreign material which has entered it. They also attest that infection can spread from the nose to the tonsils, not via the lymph channels as is usually assumed, but by the venous route. This is possible because of the cavernous plexus of the nasal conchae which bring the venous blood so close to the exposed surface that infectious agents can enter it following the slightest injury of the protective covering.

EDITOR'S ABSTRACT.

TONSILLECTOMY IN INTERNAL DISEASES AND THE PROBLEM OF ORAL SEPSIS. L. POLEDNAK and J. HORAK, Sborn. lék. **36**:19 (July 11) 1930.

The author reviews the literature in detail and cites tables and classifications of diseases directly and indirectly associated with oral sepsis. In his series 114 patients were watched for a period of eight years. The effect of tonsillectomy and dental extraction was checked in rheumatism, heart disease, nephritis, septicemia, nervous diseases, bronchial asthma, disease of the thyroid gland and anemia. Operation was carried out only in those cases in which both the internal and the local indications warranted operation. The oral treatment was usually radical, that is, tonsillectomy and extraction. All local symptoms disappeared; generally the best results were obtained in acute rheumatic conditions of the joints, rheumatic disease of the heart, oral sepsis and secondary nephritis.

STULIK, Chicago. [AM. J. DIS. CHILD.]

Larynx

ACUTE NONDIPHTHERITIC LARYNGEAL OBSTRUCTION: A REPORT OF FIFTY-ONE CASES TREATED AT THE HARTFORD ISOLATION HOSPITAL DURING THE PAST SIX YEARS. C. L. THENEBE, New England J. Med. **207**:740 (Oct. 27) 1932.

The pathologic process embodies hyperemia and edema of the involved region. The edema is often of a rapidly increasing type. A period of sixteen hours from the onset to death has been observed. An exudate or membrane is only rarely present. The fat child with a short neck seems to be more susceptible to this infection. The voice sounds are usually present.

Fifty-one patients with this condition were admitted to the hospital; thirty-eight were males and thirteen females. The distribution according to age was as follows: from 8 months to 2 years of age, twenty patients; from 2 to 5 years fourteen; from 5 to 10 years, thirteen; from 10 to 20 years of age, three; above 20 years, one. The temperature on admission varied from 100 to 105 F. Six patients were admitted on the first day of illness, with one death; eighteen on the second day, with two deaths; eighteen on the third day, with two deaths, and nine on the fourth day or over, with two deaths.

In twenty-three cases in which intubation was not performed, there was no mortality; in four cases in which intubation was performed without tracheotomy, one death occurred (a mortality of 25 per cent); in twenty-one cases in which intubation and tracheotomy were performed, five deaths occurred (a mortality of 23.8 per cent); in three cases in which tracheotomy was performed without intubation, one death occurred (a mortality of 33 per cent). The total mortality of the entire group was 13.7 per cent. The total mortality of the intubated and tracheotomized patients was 25 per cent.

Analysis of the age in the fatal cases showed: 9 months, one case; 13 months, two cases, and 14 months, 21 months, 3 years, and 7 years, one case, respectively.

Tracheotomy should be performed early before the child is exhausted from prolonged dyspnea.

GENGENBACH, Denver. [AM. J. DIS. CHILD.]

FRACTURES OF THE LARYNX BY DIRECT VIOLENCE. PIÉDLIÈVRE, DESOILLE and ROUSSEL, Ann. de méd. lég. **8**:562, 1932.

Fractures of the larynx due to hanging and strangulation with a cord and with the hand have been studied systematically, but those caused by direct trauma have not been carefully studied. A case is reported of a man struck on the neck by a blow of the fist; grave symptoms of asphyxia developed, and he died several hours afterward. At autopsy edema of the glottis and a bloody infiltration about the larynx were found, also the following fractures: (1) a median and vertical fracture of the thyroid cartilage, (2) a fracture of the left posterior portion of the cricoid cartilage and (3) a fracture of the right great horn of the hyoid bone. A review of the literature revealed that fracture of the cricoid cartilage is relatively infrequent, while fracture of the thyroid cartilage is relatively frequent. Fractures of the arytenoid cartilages, hyoid bone and some of the first rings of the trachea are exceptional. The mechanisms of the fractures are reviewed. Age and ossification increase the liability to fracture. Death occurs rapidly. Fractures of the larynx from direct violence should not be overlooked in efforts to find the cause of death.

THATCHER, Little Rock, Ark. [ARCH. PATH.]

LARYNGOSTENOSIS AND GLYCOSURIA IN A CHILD. MARTIN STRÜMPEL, Deutsche med. Wchnschr. **58**:1722 (Oct. 28) 1932.

In 1900 Leichtenstern called attention to the fact that in diabetes the vocal cords might get dry, and that if this condition persisted for a while inflammatory processes in the mucous membrane of the larynx might follow.

The author describes the case of a boy, 8 years of age, who was never ill before and in whom, in the course of six hours, dyspnea and laryngospasm suddenly developed to such an extent that tracheotomy was indicated. Laryngoscopic examination revealed that the epiglottis and trachea were swollen and red. In the course of the complete examination, 1.6 per cent sugar was found in the urine. Tests for acetone and diacetic acid were positive. After tracheotomy, proper diet and insulin, the child made an uneventful recovery and went home, having a regular diet without insulin. When the child was seen again, five months later, he was in good health, without any evidence of diabetes mellitus. Therefore, the conclusion of the author is that the laryngostenosis was not the complication of a true diabetes mellitus but the result of a general infection, which also damaged the pancreas or kidney and caused a secondary glycosuria. Both the laryngostenosis and the glycosuria were transient and ceased after the infection stopped.

KERESZTURI, New York. [AM. J. DIS. CHILD.]

CONGENITAL MALDEVELOPMENT OF THE LARYNX. ANNELISE ROHRMANN, Virchows Arch. f. path. Anat. **283**:304, 1932.

The anomaly occurred in a new-born girl with multiple malformations, and consisted in rudimentary development of the epiglottis; in place of the latter there was a group of cartilaginous nodules.

SCHULTZ, Evanston, Ill. [ARCH. PATH.]

Nose

BACTERIA CAUSING ACUTE CORYZA. L. HOYLE, J. Path. & Bact. **35**:817, 1932.

The results of extensive observation of the relation between appearances of Bacillus influenzae and pneumococci in the upper respiratory tract and the occurrence of attacks of acute coryza suggest that many of the more severe attacks of coryza may represent infections by these organisms.

AUTHOR'S SUMMARY. [ARCH. PATH.]

THE SUBMUCOUS RESECTION OF THE NASAL SEPTUM IN CHILDREN. MACLEOD YEARSLEY, Practitioner **129**:581 (Nov.) 1932.

The author presents a series of seven cases, the patients including one girl and six boys ranging in age from 10 to 14 years, in which he did a submucous resection of the nasal septum. The indications for operation were two or more of the following complaints: chronic catarrh, marked nasal obstruction, night terrors, increasing deafness, frequent colds, mouth breathing and asthma.

One of the patients was seen eight years later, and two were seen ten years later. The author points out that the scar tissue forming the new nasal septums in these three cases was as hard and as resistant as bone.

Excellent results were obtained in all cases, and from his experience the author believes that the operation is a valuable and justifiable procedure in children.

SAPPINGTON, San Francisco. [AM. J. DIS. CHILD.]

NASAL INSTILLATIONS IN INFANCY (FATAL MISTOL INTOXICATION). HERMANN BRÜNING, Arch. f. Kinderh. **93**:102 (April 10) 1931.

Several instances are cited from the literature in which dangerous and alarming results followed the use of camphor and menthol in nose drops in infancy. The author reports the case of a 6½ month old infant who after receiving mistol by nasal instillation for nine weeks showed signs of chronic poisoning and finally died. If nose drops are used in infants it is advisable to use only indifferent fluids, such as plain oil, which can do no harm. If drugs are incorporated into the nasal medication, it is advisable to specify the exact dose and the length of time administration should be continued.

LEMASTER, Evanston, Ill. [AM. J. DIS. CHILD.]

INTRANASAL PATHOLOGIC HISTOLOGY. K. FUCHS, Monatschr. f. Ohrenh. **66**:1077 (Sept.); 1215 (Oct.); 1362 (Nov.) 1932.

Fuchs discusses intranasal pathologic histology on the basis of a study of eight necropsy specimens. Special attention is given to pathologic alterations of the septum. After a discussion of the pathogenesis, etiology and histology of deviations of the septum, he discusses the histologic findings in protuberance of the septum produced by the sphenoidal process, thickening and deviation of the nasal septum by a paraseptal cartilage, deviation of the septum after surgical intervention, healed fracture of the cartilaginous septum and the appearance of lipoid tissue in the septum. Other intranasal alterations discussed include: rhinitis papillaris, inflammatory atrophy of the nasal turbinals, synechia of the septum and lateral nasal wall and deposits of calcium in the mucosa of the sphenoid sinus.

EDITOR'S ABSTRACT.

CONGENITAL MALDEVELOPMENTS OF THE NOSE. B. LANG, Virchows Arch. f. path. Anat. **285**:93, 1932.

Two unusual examples of nasal maldevelopment are described, one of proboscis lateralis and one of Kundrat's cebocephalic form of arhinencephaly. In each there were multiple malformations in addition to the nasal one, to which special attention is given. In the case of proboscis lateralis, the left half of the nose was normally formed. The right half was represented by a short, sessile structure attached at the inner canthus of the right eye. The right nasal bone and the right nasal cavity were absent. There was a coloboma of the right lower eyelid. The intermaxillary bone was absent. In the case of cebocephaly a rudimentary nose with a single nostril had been formed. The following structures had not been formed: the horizontal and vertical plates of the ethmoid, the small wings of the sphenoid, the optic foramina, the vomer, the lacrimal bones, the intermaxillary bone and the nasal cavities. The frontal lobes of the brain were fused in the midline, with maldevelopment of the olfactory lobes and other parts of the brain. The endogenous and exogenous factors that may have a part in the genesis of such maldevelopments, and the period of embryologic development at which the latter may arise, are discussed.

O. T. SCHULTZ. [ARCH. PATH.]

DOUBLE MIDDLE TURBINATE. RUDOLPH MICHL, Časop. lék. česk. **70**:1472 (Oct. 30) 1931.

The author reports a case of complete duplication of the right middle turbinate bone. The question of embryology is discussed briefly.

STULIK, Chicago. [AM. J. DIS. CHILD.]

Society Transactions

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Regular Meeting, Jan. 4, 1933

CHARLES B. YOUNGER, M.D., *President, in the Chair*

RIGHT-SIDED RETRO-ESOPHAGEAL AORTA (PERSISTENCE OF THE RIGHT AORTIC ARCH): A NEW CLINICAL AND ROENTGEN PICTURE. DR. AARON ARKIN (by invitation).

A new form of aortic anomaly was presented. It consists of a total persistence of the right aortic arch, together with a persistence of the left aortic arch in the form of the left subclavian artery, a short obliterated vessel and a diverticulum of the aorta; in other words, a persistence of both aortic arches. The right arch forms the aorta. The left arch forms the beginning portion of the left subclavian artery, the obliterated vessel (isthmus stenosis) and the aortic diverticulum (left descending dorsal aortic root).

The patient, a man, aged 50, was examined by Dr. Arkin at the Wenckebach Clinic, Vienna. He presented the following roentgen appearance: 1. There was a shadow to the right of the sternum, running upward to the region of the head of the right clavicle, with a distinct systolic pulsation. 2. There was an absence of the normal aortic knob to the left of the sternum. 3. The right oblique position showed the aortic knob behind the trachea and esophagus; both of these were displaced forward and to the left. 4. The retro-esophageal aortic shadow contained a round shadow representing the diverticulum. Dr. Arkin made a diagnosis of persistence of the right aortic arch which ran over the right bronchus and behind the trachea and esophagus. This was confirmed by the autopsy.

At autopsy, a total persistence of the right aortic arch which ran over the right main bronchus and behind the trachea and esophagus was found. At the place where the arch went over into the descending aorta was a diverticulum 1.8 cm. in diameter and 1.5 cm. long. From this an obliterated vessel extended to the pulmonary artery (ductus botalli) and a second short obliterated vessel to the left subclavian artery. The first branch of the aorta was a left innominate artery, which divided into a left subclavian and a left common carotid. The second branch was the right common carotid, and the third the right subclavian artery. The heart revealed no anomalies.

There is here a total persistence of the right aortic arch as occurs normally in birds. As in most cases of right-sided aorta, so also in the case reported, the left ductus botalli persisted and not the right. The left fourth branchial artery formed the left subclavian and the short obliterated vessel, and the left descending dorsal aortic root persisted in the form of the aortic diverticulum. This form of right-sided aorta differs from those previously described because of its retro-esophageal course. The persistence also of the left arch explains this difference. Dr. Arkin had seen six cases to the time of presentation.

A new roentgen picture is thus established and confirmed post mortem, which enables one to diagnose this interesting anomaly with certainty in vivo. Five additional cases of the anomaly have been diagnosed by Dr. Arkin in patients without cardiac symptoms. They presented the identical roentgen findings described. A second case was diagnosed and confirmed at autopsy.

These cases are of embryologic interest because they represent a reversion to an avian type of aorta in man, or rather to the reptilian type of double aortic arch. They also illustrate the fact that right-sided aorta occurs without any cardiac anomalies.

These cases of double aortic arch are of special interest to bronchoscopists, because of the displacement of the trachea and esophagus by the aortic arch, which lies behind both instead of in front. Serious injury might result from the passage of a bronchoscope or an esophagoscope. These cases emphasize the importance of a careful fluoroscopic examination, including examination of the esophagus, before every bronchoscopic or esophagoscopic examination.

DISCUSSION

Dr. E. L. Jenkinson (by invitation): I do not think that Dr. Arkin left much to be said. When he returned from Germany he came to see me, and we became interested in this anomaly. There is probably no other system in the body that is more prone to have anomalies than the vascular system. Some are of interest only to the anatomist, while others are of great practical value. As Dr. Arkin pointed out, this anomaly is probably of great interest to the esophagoscopist and bronchoscopist, and that means more work for the radiologist, in having fluoroscopy performed before bronchoscopy. In our work we have not been as fortunate as Dr. Arkin is seeing these cases. Over a period of a good many years, on doing a great many examinations of the stomach and esophagus, I have seen only one case, and that I saw with my brother. That takes in a period of sixteen or seventeen years and means thirty-five or forty thousand gastro-intestinal examinations. Possibly we are not recognizing the anomaly, but from the one I saw with my brother I do not think that it is difficult to identify. I have gone over the work of Brigham, and among fourteen thousand cadavers dissected, he found only one case of right-sided aorta. Abbott found fourteen among a thousand cases. Probably many of us do not identify the anomaly, and I do not doubt that the cases are more common than we have been led to believe.

In the one case I saw the patient came in for a general gastro-intestinal examination. I observed no difficulty in his swallowing, no cyanosis and practically no symptoms. The condition was discovered accidentally. From an anteroposterior film I am not sure that one would be able to say that there was a double aorta. We find changes in the position of the trachea and the knob in a number of conditions, and from the anteroposterior view we should not make the diagnosis. It is essential that one have the right oblique plate and use barium sulphate for complete examination.

Dr. Aaron Arkin: I might add, first, that these anomalies of the aorta are not, as a rule, associated with cardiac anomalies. The embryologic development of the aorta is more or less independent of the cardiac development. Second, these anomalies do not present symptoms unless there is an aneurysm of the aorta or syphilitic aortitis with compression of the esophagus and dysphagia. Third, these anomalies, I believe, are more common than the pathologists and roentgenologists realize. Before I called the attention of our pathologist to this condition one case was missed at autopsy, because the usual plan is to cut open the esophagus and cut the arch of the aorta without seeing its exact location. On roentgen examination of the stomach, the esophagus should always be carefully examined from the pharynx to the stomach. Otherwise, the characteristic displacement first described by me in this anomaly will be overlooked.

Not only can the diagnosis be made with the anteroposterior view by the presence of the displacement of the trachea and esophagus slightly to the left and the presence of the pulsating shadow that runs upward on the right side toward the head of the right clavicle, but it can be made by the fact that the semicircular displacement of the esophagus is visible on rotating the patient. My last case I diagnosed clinically by finding pulsation in the second right intercostal space and the supraclavicular fossa, finding the displacement of the esophagus and the dysphagia, which really was due to carcinoma of the cardia of the stomach. The diagnosis was confirmed by fluoroscopic examination, and you saw the photographs of the specimen.

ILLUSTRATED HISTORY OF THE DEVELOPMENT OF PERORAL ENDOSCOPY. DR. S. J. PEARLMAN.

To Phillip Bozzini of Frankfort is ascribed the idea of first attempting to illumine the various cavities of the body. In 1805-1807 he first described his "light carrier," illuminated by a candle and adjusted to a number of different speculae suitable to the vagina, rectum, larynx and other organs. Bozzini's efforts met with much criticism, and it is questionable how really practical his device was.

Following Bozzini came a number of other workers devoted to the same idea of inspecting the body cavities by means of illuminating devices. It must be said that the chief interest of most of these men at first was not directed toward the nose and throat, but rather toward the urethra, bladder, rectum and vagina.

Among the most important were Fisher (1845), Ségalas (1827), Desormeaux (1865) and Stoerk and Semeleder (1862). Of these, perhaps the most important was Desormeaux, with whose lighting apparatus and a long rigid tube Kussmaul is said to have done the first esophagoscopy.

The various means for providing light, such as candles, gasogen and platinum loops, were largely ineffectual and were replaced by electric bulbs shortly after Edison's epoch-making invention (about 1886). Mikulicz with Leiter's aid devised an instrument consisting of a rigid tube and lighted by a handle containing a glowing platinum wire. This handle had to carry a water-cooling device. With the practicality of examining the esophagus by means of a rigid tube well established by Mikulicz, progress was steady.

The trachea was also investigated by various workers; chiefly among them were Türck, von Schrötter, Stoerk and Pieniażek (1888). At first the examinations were made by way of a tracheotomy opening. After Kirstein was responsible for first examining the larynx by means of a spatula in a practical way, a number of workers, more by accident than by intent, stumbled into the trachea while attempting esophagoscopy, and it was Killian's (1897) service to show that tracheal examinations perorally were possible as a routine measure.

For anesthesia, at first, ether or morphine was used. With the advent of cocaine Hacker, Stoerk, Mikulicz and their followers, who labored so hard to establish the technic, found many of the difficulties of anesthesia solved.

Following the pioneers came those who perfected instruments and technic. Among them were Brünings, Eicken, Guizes, Jackson and others.

Vincenz Mueller and Company (Chicago) have a historical exhibit which shows in almost unbroken line the development of instruments intended to examine and permit operative work in the larynx, tracheobronchial tree and esophagus. Mr. Mueller demonstrated these instruments in connection with the paper.

DISCUSSION

DR. E. W. HAGENS: I wish to repeat a few of the things that Dr. Pearlman stated, but more briefly, so that we can get in order the development of the endoscopic procedures.

In 1868, Kussmaul performed the first real esophagoscopy. In 1881, Mikulicz examined the stomach. In 1891, Gottstein used local anesthesia for the first time, and also used the metal tube for the first time. In 1897, Kirstein described the first direct laryngoscopy, and in 1897, Killian did the first real bronchoscopy, removing a foreign body from the bronchus. In 1902, Einhorn, of this country, developed distal illumination for the first time.

In regard to some of the men in Chicago, I wish to mention a little more in detail the developments occurring in our own locality. Ingalls was one of the first men in this territory to use this work, reporting a case in 1898. In his textbook of 1900, he does not mention direct laryngoscopy or endoscopy, however. Following the first report he recorded six other articles, the last in 1916. He was the pioneer in this locality and was considered the leading man in the middle west. He also devised several instruments, and in the earlier years used a Killian

esophagoscope with a separate distal light-carrier. He developed the separate light-carrier, which was placed in the lumen of the tube in 1904, but in 1905, following Jackson, he improved on this by having the light-carrier inserted in a crevice in the wall of the tube, differing from Jackson, who had it entirely in the wall of the tube. This light-carrier was not covered on the lumen side of the tube, so it could be removed and the crevice cleansed. So we find Ingalls, a Chicago man, as one of the pioneers of America in developing this work at a time when Jackson also was getting started. Jackson, in 1907, in his book stated that other men were perhaps more qualified than he, but that he was writing the book because no one had ever published the work in book form so that others could learn it. The literature up to that time showed about three hundred and fifty-eight articles on the subject, but none in book form. This shows that a good deal of work had been done even at that time.

Another man to be mentioned is Friedberg, who was an assistant to Ingalls. Starting in 1900, Friedberg did a great deal of bronchoscopic work and assisted Ingalls. He did not publish much, but in 1911 published an article on bronchoscopy. There are few references in the literature, but he did a great deal of work here in Chicago and was well known in the middle west. He was a member of the American Peroral Endoscopic Association.

Others in Chicago, for example, Dr. Boot who was with us until recently, and Dr. McGinnis, have done this work, but have not published it to a great extent.

In regard to the mortality, it is stated that external esophagotomy for foreign bodies had a mortality of from 25 to 40 per cent. With our endoscopic procedures this has been reduced to 2 per cent or less. I think that the mortality depends on several factors. Certainly it would depend on the operator's ability and qualifications. If he is well qualified, the mortality rate will be low. Fatalities in work with foreign bodies are commonly due to the patient's delay in coming to the physician, thus allowing complications to develop. Another important factor is the tampering that is done by physicians or surgeons who use indirect means and push the foreign body down, or remove it and produce complications that prove fatal. If the patient is properly cared for, the mortality should be very low in cases of foreign bodies. In diseased conditions, the mortality depends on the degree of the disease. The development of endoscopic measures has been a great improvement over what was done before they were brought out.

A paper such as Dr. Pearlman's is of great value; it is of value to the older men, for while they have watched the development of the work it is not possible to see the whole subject in perspective except in a review over the last hundred years. It is of value to the younger men, for they are inclined to think either that the procedure has always been known or that it is entirely new. A review such as this shows that the things were known a number of years ago, and it will save the younger men from thinking that they are inventing something. It is valuable to all of us, and I appreciate the paper very much indeed.

DR. CHARLES B. YOUNGER: I believe that in relating the history of endoscopy so far as Chicago is concerned, Dr. Casselberry should not be overlooked. He was contemporaneous with Ingalls and did splendid work along this line. He was ingenious, and some of the esophageal and bronchoscopic instruments he devised are still in use.

FOREIGN BODIES IN THE FOOD AND AIR PASSAGES. DR. C. H. CHRISTOPH.

Dr. Christoph showed thirty x-ray pictures of foreign bodies in the esophagus, larynx and tracheobronchial tree. Ten of these were opened safety-pins in the esophagus in children ranging from 6 months to 7 years of age. The remaining foreign bodies consisted of coins, nails and miscellaneous objects. A simple method of extracting open safety-pins was given. The difficulties encountered in the diagnosis and removal of foreign bodies in the respiratory passages, especially the nonopaque objects, were discussed.

DISCUSSION

DR. JOHN DELPH: There is little that I can add, because Dr. Christoph has covered the field very well, but there are two or three things I should like to mention.

Early tracheotomy following the removal of vegetable foreign bodies is important in some cases, as has been learned from experience. I had an unhappy experience recently because I did not perform a tracheotomy early enough and follow it with the Jackson method of dropping down physiologic solution of sodium chloride. I watched carefully and thought that the child was doing very well, but it choked suddenly and drowned in its own secretion.

There are some interesting things in work with foreign bodies. On one occasion I saw a patient in an asylum for the mentally defective who had taken a spoon, bent it double and pushed it into the esophagus. An attempt at removal was made, and I was asked to assist. I could not get the spoon out for it would stick, and we decided that the best thing to do was push it into the stomach, which we did, and it was then removed by gastrostomy. The man made an uneventful recovery.

It is also surprising to see the various types of objects that get into the air passages. I have seen irregularly shaped objects that I thought could not pass, and I have seen several heavy objects that happened to hit the epiglottis in such a way as to pass into the trachea.

One has to be careful in removing foreign bodies so that they will not drop back into the airways. If they do not drop back into the airways, they sometimes go into the esophagus during an attempt at removal.

Regular Meeting, Feb. 6, 1933

CHARLES B. YOUNGER, M.D., *President*

(Program furnished by the University of Illinois, School of Medicine.)

ARTIFICIAL PRODUCTION OF SYNCOPE. DR. J. W. HARNED (by invitation).

This demonstration of artificial syncope was first presented at the University of Illinois following a paper by Dr. J. Gordon Wilson on laryngeal vertigo, with the idea that artificial syncope might have some relationship to laryngeal syncope.

The procedure to produce the condition is not difficult. The subject is instructed to take three deep breaths with the arms held straight out; as the last breath is held, the operator stands behind the patient and encircles the chest with both arms. He then lifts him from the floor, applying moderate pressure to the chest. Fainting occurs in from ten to twenty seconds; the arms drop to the side, air is exhaled from the lungs with a sigh, and the subject falls languid to the floor.

The blood pressure is found to drop about 10 points, and the pulse remains the same. The patient's face is flushed and congested at first, but later becomes pale.

Respiration does not begin for from ten to thirty seconds, and then, as convulsive movements of the arms and legs occur, breathing is resumed.

Five theories have been advanced regarding the cause of this syncope: (1) artificial anemia of the brain, (2) a supersaturation of carbon dioxide in the blood stream, (3) a supersaturation of oxygen in the blood stream, (4) laryngeal epilepsy or laryngeal syncope and (5) vasovagal syncope. I think the last is the most appropriate theory.

It is of interest to note that after a full meal syncope cannot be produced except under unusual circumstances. (This was demonstrated on a laboratory technician who had dined and on whom the method failed to produce syncope. In a second demonstration syncope was readily produced in a young lady who had not dined.)

DISCUSSION

DR. J. GORDON WILSON: Dr. Harned's demonstration of artificial syncope shows the results that may follow a slight increase of intrapulmonary pressure. There was no marked blueness of the face, only a general duskiness, and certainly no pallor. There was a quick return to consciousness, and one could observe a fleeting mental confusion. It is well known that increase of intrapulmonary pressure can be produced by holding the breath (closure of the glottis) and by pressure on the chest, and that it may be followed by an uncomfortable feeling in the head, nausea, vertigo and mental confusion. In such cases the blood pressure falls rapidly, for instance, from a normal of 114 to 70, quickly followed by a rise, even to 140. There is a diminution in the amount of blood reaching the left ventricle. There is a varying amount of cerebral vascular disturbance accounting for the cerebral symptoms. In addition to the intrapulmonary pressure, there is a reflex to the medulla along the vagus from the afferent respiratory nerves.

Syncope resulting from intrapulmonary pressure presents symptoms similar to those which may accompany laryngeal vertigo. But in laryngeal vertigo the essential and primary factor is the closure of the glottis associated with the vagus reflex. If the closure is prolonged, it may easily pass to a circulatory disturbance with symptoms of intrapulmonary pressure. In typical cases of laryngeal vertigo, there is no mental confusion discernible.

Artificial syncope is not without danger. It may be harmful to the healthy and prove disastrous if there is any cardiovascular disease.

The question has been asked, Are breath-holding spells related to laryngeal vertigo?

Both are primarily of reflex nervous origin. Breath-holding spells are due to spasm of the glottis (adductor spasm); laryngeal vertigo occurs in adults and is ushered in by closure of the glottis. In both, subsequent to the closure, respiratory and circulatory disturbances may follow, varying in degree with the duration of the spasm. The child often shows greater involvement of the nervous system than the adult, which may be accounted for by age. In neither can a local lesion be demonstrated in the larynx; in both, recovery is rapid and complete.

Syncope as here demonstrated is due to increased intrapulmonary pressure; it is different in its causation, though often presenting somewhat similar symptoms.

DR. T. C. GALLOWAY: What is the relation of this condition to the tabetic crises of the larynx?

DR. J. W. HARNED: I do not know that I can answer that. Dr. Wilson spoke of the possibility of accidents in this connection. Since I took up the study of artificial syncope in 1922, I think I have probably caused about six hundred persons to faint, and in that span of years I have never had an accident except on one occasion. There was a young man in the university who wished to have the experiment attempted on him. He fainted immediately, but said he did not think it could be done again, that he could prevent himself from fainting. There were about three minutes between the two attempts. The first time the boy was unconscious about twenty seconds, but the second time the unconsciousness lasted a minute and a quarter. I have seen this done two or three times since. If a fainting spell is produced within half an hour of the original faint, the subject will remain asleep much longer than the first time. A physician saw this experiment performed in the hospital one morning; that afternoon a man came into his office who had injured his hand, and it was necessary to take some stitches. The doctor decided to try this procedure and took three stitches in the hand before the patient awoke.

I have heard of one accident in one of the colleges where the experiment was tried on a young man who had myocarditis. Before I try it I always check up to see if the patient has hypertension or any cardiac disease.

DR. FRANK NOVAK, JR.: What changes in the pulse rate occur?

DR. J. W. HARNED: The pulse rates are slow. On the subjects we have checked, the rate has dropped to 65 or 60, a drop of from 10 to 15 points, but the beats were of full volume. We have had more than a 10 degree drop in the blood pressure.

VASOMOTOR REACTIONS. DR. IRWIN G. SPIESMAN from the ear, nose and throat department, University of Illinois, School of Medicine (by invitation).

Our experimental studies on the common and repeated cold in the head were begun three years ago. The objective was to pursue further the researches made on the vasomotor responses of the upper respiratory tract to thermal stimuli applied to the cutaneous surface. The experiments were conducted in a regular laboratory in which drafts were minimized. The findings are based on 164 experiments. The thermo-electric method was employed with a thermopile electrode applied to the mucosa of the nose and the electromotive force measured with a potentiometer. Cold air sprays and aluminum cups, varying in diameter from 4 to 19 cm., partly filled with chipped ice, were used as cold stimuli. The cups were applied to the upper and lower dorsal vertebrae and to the lumbosacral regions of the back, to the upper and lower anterior portion of the chest, to the upper and lower part of the abdomen, and to the feet. These experiments were performed on eight normal men and on thirty-one persons with hyperesthetic rhinitis, including three children. The purpose was to determine the normal and abnormal response of the mucosa of the upper respiratory tract to a cold stimulus applied to the cutaneous surface.

In the normal subjects the findings were as follows: The smallest cold stimulus applied to the cutaneous surface effecting a reflex lowering of the temperature of the respiratory mucous membrane was a 4 cm. ice cup. The response to a short circumscribed cold stimulus (two minutes) was first a lowering of the temperature of the mucous membrane, then a rise and a gradual return to the initial temperature. The response to a continuous circumscribed cold stimulus (ten minutes), whether to one cutaneous area or to numerous cutaneous areas applied in regular succession, i.e., when the reaction was completed in one area, the cold stimulus was immediately removed to another, was first a lowering of the temperature of the mucous membrane, then a gradual rise, stopping short in most of the experiments of reaching the initial temperature by from 0.18 to 0.36 C.

With a continuous cold stimulus, the thermal endings in the skin gradually adapted themselves to this stimulus. This process of adaptation is associated, to an extent, with the phenomenon of fatigue. Further experimentation is being conducted to determine whether this continuous cold stimulus which produces a reflex vasoconstriction, first of maximal and then of minimal degree, is a phenomenon of adaptation or fatigue.

It was further found in normal persons that a cold stimulus to an anesthetized cutaneous area in which pain was removed produced a reflex lowering of the temperature of the mucous membrane. Since thermal impulses follow a course similar to pain, this proved that pain is not a factor causing vasoconstriction. Moreover, changes in the temperature of the mucosa of the upper respiratory tract to cold stimuli were proved to be caused by a reflex change in the vasomotor tone of the vessels supplying the mucosa. A fall in temperature indicated a vasoconstriction; a rise, a vasodilatation.

These findings are contrary to earlier assumptions that chilling of the cutaneous surface was associated with congestion of the mucous membrane of the nose and throat, and that this congestion was favorable to bacterial invasion.

In abnormal subjects, patients with a cold in the head, it was found that during and for several days following a cold a period of vasomotor suspense existed; i. e., a cold stimulus applied to the cutaneous surface produced no vasomotor response in the mucous membrane. The suspense of the vasomotor reaction is unquestionably of a local effect, within the mucosa, produced by the toxic products of the infection on the vasomotor endings in the mucous membrane.

The persons with hyperesthetic rhinitis manifested erratic vasomotor reactions. A cold stimulus applied on the cutaneous surface produced, frequently, a reflex rise instead of a drop, and a hot stimulus produced a lowering instead of a rise in the temperature of the mucous membrane.

DISCUSSION

DR. M. B. VISSCHER, professor of physiology: It is particularly fortunate that this type of investigation is being carried on by men in otolaryngologic

work, because the solution of some difficult problems in connection with susceptibility to disease lies in the application of the methods of physiology to these problems. We have been impressed for a long time with the importance of exposure to infection in the causation of disease, but have largely neglected the more important problem, so far as the individual is concerned, of the individual's susceptibility to disease. The things that determine individual susceptibility are of the sort about which Dr. Spiesman has been talking. It is often forgotten that changes in the physiologic condition allow organisms already present to grow. So far as the individual is concerned, this fact is more important than the question of infection itself. In this instance, the importance of the findings of Dr. Spiesman, and others who have worked in this direction, may not be apparent on the face of them. In saying that there is a change in temperature of 2, 3 or 4 degrees in the mucous membrane of the nose and throat, it is not immediately apparent that there is a marked change in metabolic conditions, but there is really a marked alteration in blood flow that produces such a fall of temperature. A fall of even 1 or 2 degrees may mean that there is an absolute anemia of the mucosa and consequently a lack of oxygen which is extremely favorable for the growth of a large variety of pathogenic organisms. The changing resistance to infection with alterations in the supply of oxygen to the tissue is the factor of importance which Dr. Spiesman has been studying. To show that the normal reflex response to a change in the temperature of the skin effects a change in the nasal mucosa of the sort that has been described seems to be an important step in the analysis of the mechanism of production of infection of the upper respiratory tract.

DR. LLOYD ARNOLD, professor of bacteriology: The report of the problem we have just heard has been presented a little differently from that of the bacteriologist. I should say it is a combination of bacteriology and physiology applied to clinical medicine. We have been studying body surfaces for ten or twelve years, including the skin, the oral cavity, the nasal and respiratory tracts, the digestive tract and, to some extent, the genito-urinary tract. We have been trying to find out how bacteria adapt themselves to these body surfaces. We have a bacterial flora living on each body surface that is distinctive of this area. We not only have certain strains of bacteria but we also have the problem of the relative density of the bacterial population on body surfaces. These factors must be controlled by something, and we have been trying to study this controlling mechanism. Most of our work has been confined to the gastro-intestinal tract, but we have also studied in some detail the respiratory tract.

Returning now to Dr. Spiesman's problem, we have been trying to determine whether the mucosa of the upper respiratory tract with its capillary beds reacted similarly to the skin area or to the gastro-intestinal tract. Stated briefly, we have found that the respiratory tract reacts similarly to the skin. That is, vasoconstriction of the skin is immediately followed by vasoconstriction of the respiratory tract and vice versa. The gastro-intestinal tract reacts in the opposite direction to the skin.

We have three lines of evidence supporting Dr. Spiesman's work in our experimental studies in which dogs were used. One line of evidence is similar to that presented by Dr. Spiesman; namely, the temperature of the mucosa of the respiratory tract, from the larynx to the small bronchi, follows that of the skin. We have used the capillary microscope to study the size and shape of the capillaries in the trachea. These capillaries increase in size, and the number in a given field increases when the temperature of the skin is elevated by hot packs. The opposite picture presents itself when the skin is chilled. The third line of evidence consists of white blood cell counts made simultaneously from the skin and the tracheal mucosa. When the temperature of the skin is elevated by hot packs, there is an increase in the number of leukocytes per cubic millimeter of blood in both the skin and the tracheal mucosa. The opposite occurs when the skin is chilled.

We have found in our preliminary experiments that a body surface is most permeable for bacteria during sudden contractions of the capillary plexus. This holds not only for the respiratory tract but also for the kidney. I consider Dr.

Spiesman's work a worth-while contribution to the better understanding of disturbances of the upper respiratory tract and their sequelae.

DR. I. HARRISON TUMPEER: Those of us who are interested in allergy will applaud Dr. Spiesman's effort to study the relationship of the temperature of the skin to the temperature of the nasal mucosa.

No proper presentation of this subject can be given without proper orientation. It must be apparent to rhinologists and laryngologists that the observations reported this evening owe their sole importance to the relationship of the temperatures of skin and mucous membranes to the clinical fields of hyperesthetic rhinitis and "colds" in general. The essayist spent a lot of time with references from the foreign literature. In fact, he had to leave his summary to the final discussion. There can be no excuse for leaving out the name of Duke and other Americans in an introduction which took so long and emphasized foreign writers. This piece of pure laboratory observation owes its being to the fact that clinicians have long recognized that exposure to cold often causes nasal symptoms. To discuss only Duke and his contributions would take too long. Suffice it to say that he has contributed an entire chapter to allergy, demonstrating that heat or cold applied to the skin of different persons causes stuffing of the nose and even wheezing. He has even demonstrated the quantitative relationships of heat and cold. If a man plunges his arm in cold water, râles and wheezing may develop, and if he plunges one arm in cold water and one arm in hot water this may be avoided. Without going into further detail concerning Duke's other contributions, I want to make clear that the essayist has omitted certain pertinent observations of Duke in his own paper. In the first place, it is a coincidence that the temperatures Dr. Spiesman used are the temperatures Dr. Duke has found physiologically adequate for cold stimulation, namely, from 5 to 15 C. Secondly, the essayist has merely mentioned the fact that the application of heat to the skin after the application of cold produces a greater elevation of temperature in the nasal mucosa than the application of heat in the beginning. The essayist made no attempt to elucidate this finding. On the other hand, Duke called attention to the clinical fact that heat produces symptoms particularly after cooling. For instance, nasal symptoms develop in a man when he comes from the wintry outdoors into a warm room, while this same temperature does not affect him without the preliminary cooling. Duke has commented that these persons are considered cold "sensitives," when in reality they are heat "sensitives." Thirdly, from the standpoint of clinical application, undue emphasis has been placed on the first six charts demonstrating the so-called normal reaction. Only the last chart was presented to show that heat to the skin in some persons lowers the temperature of the nasal mucosa and vice versa. This group forms the class likewise described by Duke, the group of hypersensitive subjects who present clinical symptoms.

In conclusion, the purely laboratory work presented is but a minor attempt to give laboratory sanction to only one small part of the larger field of clinical fact fully uncovered by Duke.

DR. FRANK J. NOVAK, JR.: It seems to me that in order to conduct an investigation of this type a comprehensive study of the normal must precede a study of the abnormal. If the first six charts presented had not been shown, the nature of the erratic curve could not have been interpreted or understood. I must differ with Dr. Tumpeer on this point.

I wish to ask whether in attempting to determine the normality of those persons who comprise the first five or six groups the question of the metabolic rate was at all considered. We know that this has an influence on such things as were taken up here.

DR. HARRY L. POLLOCK: The subject presented by Dr. Spiesman is interesting and important, but I was surprised that he did not mention the excellent work carried on by Undritz and Sassossow of Leningrad. By the use of cold applications to the abdomens of dogs and rabbits, these experimenters demonstrated a rather definite drop in temperature of the nasal and tonsillar mucosa. This same effect was produced by inhalation of cold air or ligation of both carotids.

The amount of decrease in temperature also is dependent on the constitutional factors of the animal.

The drop in temperature is produced chiefly through reflex vascular contraction, although the slowing in the local circulation is also a factor.

I think that investigations of this sort are important in the prevention of the diseases which are prevalent during the cold seasons of the year, since it is likely that inhalation of cold air, especially in mouth breathing, has a tendency to decrease the immunity powers of a person and thus assist in causation of anginas.

DR. I. G. SPIESMAN: The basal metabolic rate was not included in this study. Eight normal young men were used for these experiments, and we did not feel that a metabolic study was essential to the experiments. The short time allowed for this paper was insufficient to permit a comprehensive review of the literature on the subject. Undritz (*Ztschr. f. Hals-, Nasen- u. Ohrenh.* **32**:3, 1932) produced an interesting paper relating to this subject. Azzi (*Riforma med.* **37**:175, 1921) showed that the reflex lowering of the temperature of the tonsils to cold stimuli was greater than of any other tissue he studied except that of the nose.

In addition to the summary of the findings already presented in this paper, it is significant to add that the daily constitutional state of the subject, as effected by his emotional, digestive and atmospheric environment, such as changes in the temperature and the humidity, are factors altering his vasomotor reactions. The altered vasomotor state is abnormally stimulated by cold applied cutaneously and effects reflexly the mucosa of the upper respiratory tract in a prolonged state of vasoconstriction. Such a mucous membrane with a diminished blood supply—a tissue which is now not normally aerated—presents the milieu for infection.

SPONTANEOUS RUPTURE OF AN ABSCESS OF THE TEMPOROSPHENOIDAL LOBE THROUGH THE SKULL. DR. B. LAMBRAKIS (by invitation).

A man, aged 27, had been well until Oct. 22, 1932, when he complained of aching over the right side of his face and pain in the teeth on the same side. The third right molar, which was thought to be infected, was extracted, but the symptoms became progressively worse. Within five days a swelling appeared over the right temporal region.

On November 1, the patient visited my office, conplaining only of headaches. The temperature was 99 F. and the pulse rate 70. Functional examination of the right ear showed excellent cochlear response in spite of an old adhesive process. There was no evidence of any actual or previous perforation of the drum. The swelling was above the squama temporalis. It was about the size of an apple, and was very painful to touch. The diagnosis of an abscess was made.

The patient was sent to the hospital. An incision was made over the area, and a large amount of mucopurulent material was removed. The temperature at this time was 100.6 F. The swelling diminished following the removal of the pus, but the same evening reappeared.

The patient spent a restless night, complaining of headache and pain on the right side of the face. The following morning, large amounts of pus were still discharged from the incised area. A probe was inserted, which suddenly passed into a jagged opening in the skull for about 2 inches (5 cm.) into the cranial cavity, from which pus exuded. A roentgenogram determined that the probe extended directly medially from the point of entrance about 3 inches (7.6 cm.) along the middle fossa of the base of the skull and about 0.5 cm. above the base of the skull, and pointed toward the mastoid cells. The mastoid cells showed sclerosis and underdevelopment on the right side, as produced by an old mastoiditis.

On physical examination the only positive findings were a point of drainage over the right zygomatic bone, limitation in opening the mouth and a doubtful Kernig sign. The white blood count was 11,200, with 75 per cent neutrophils. A diagnosis of extradural abscess was made.

On November 8, the third day in the hospital, the patient was again operated on. A U-shaped temporal flap was made above the zygoma, and the fossa above the zygomatic bone was exposed in an area 5 cm. in diameter. The dura was covered with organized exudates and granulations with free pus appearing with

each bite of the rongeur. The area was uncovered until the healthy dura was reached. A culture of the pus showed Staphylococcus albus. The spinal fluid cell count was 40.

During the next five days the patient was rational, complaining of pain on the right side. The temperature was of the septic type, varying between 97.6 and 102 F. with a definite rise in the afternoon. The leukocyte count dropped from 11,200 to 9,200.

On the seventh postoperative day the temperature rose to 103.2 F., and the leukocyte count to 17,400. The patient's mind became hazy; there was difficulty in swallowing, some vomiting and hiccups. On the following day the patient was hard to arouse; the hiccups were more severe, involuntary urination occurred, the pupils were unequal, and there was slight ankle clonus.

On the ninth and tenth postoperative days the patient was unconscious most of the time; the pupils did not react to light, and no Hoffmann or Babinski sign was elicited. No rigidity of the neck or Kernig sign was present.

On the eleventh postoperative day, a flaccid paralysis of the left upper and lower extremity was present, as well as a left foot drop and bilateral ankle clonus.

On the sixteenth postoperative day, an operation was performed on the right mastoid, which revealed that the mastoid cells were completely destroyed, the interior of the mastoid being converted into a cavity which included the antrum. This cavity did not contain pus or granulations. The bone was removed to make a continuous connection between the previous operative area and the mastoid cavity, pus being found covering the dura all along the new tract.

On the nineteenth postoperative day, the point of a needle was inserted into the bulging area of the dura mater behind the right ear, and 5 cc. of pus was aspirated. The same evening the blunt end of a hemostat was inserted, and thick, foul smelling pus exuded from the brain continuously for a period of fifty minutes.

The following day a small quantity of pus still exuded from the brain. The patient sank rapidly and died at 10:50 p. m.

Necropsy showed the dura over the right temporal lobe to be markedly thickened and indurated with a brownish-red discoloration covered with a purulent exudate over a distance of 7 by 6 cm. In the lower portion of this indurated area was an opening 12 mm. in diameter, through which exuded a creamy yellow pus. A large cavity extended throughout the entire length and width of the temporal lobe. This cavity was filled with pus and lined by a coat of organized necrotic substance. Just superior to this cavity was a smaller one similarly constructed, about 2 cm. in diameter.

Microscopic examination of the lining wall of the large abscess revealed marked fibrosis, indicating a process of many years' standing.

Tumors of the Cerebellopontile Angle: Report of Two Cases with Operative Findings. Dr. S. L. Shapiro.

The subject of tumors of the cerebellopontile angle has been thoroughly investigated, especially through the efforts of such men as Henschen and Cushing, and in our own field by Jones and Fisher. My purpose in presenting these case reports is to discuss certain clinicopathologic findings of particular significance for otologists, and to call attention to a case in which a clinical diagnosis differentiating two forms of tumors of the cerebellopontile angle was made preoperatively and later confirmed at operation and by histologic examination.

Case 1.—A woman, aged 50, was admitted on the neurologic service of the Research Hospital, University of Illinois, with a complaint of tinnitus and deafness in the right ear of four years' duration, succeeded in order by hypesthesia of the right side of the face, ataxia, headaches and failing vision.

Examination indicated involvement of the fifth nerve, incoordination of the lower extremities, a positive Romberg sign and moderate choked disk. The cochlear examination by the otologic department indicated complete deafness for all sounds on the right side and a slight impairment of the inner ear on the left.

Vestibular examination showed a spontaneous nystagmus in the direction of gaze everywhere excepting downward. With the eyes in midline, there was only an occasional horizontal twitch with the fast component sometimes to the right and at other times to the left. The nystagmus varied in intensity, rhythm and amplitude, especially on fixation to the side, and presented that appearance of ataxia which Dr. J. Gordon Wilson has so aptly termed the most characteristic feature of cerebellar ocular movements. There was a spontaneous past pointing with the right arm to the right, arm-drift reaction and gait deviation to the right with the eyes closed, and the Romberg sign was demonstrated, the patient swaying backward and to the right.

The caloric tests with the Bárány douche at 68 F. gave no response whatever for either vertical or horizontal canals after irrigation of the right eye for five minutes and a good response with a normal nystagmus, as observed in midline, following irrigation of the left ear both with the head 30 degrees forward and 60 degrees back. Past pointing was in the proper direction. Rotation tests indicated shortening of the duration of the nystagmus and vertigo to be more marked after the patient was turned to the left.

Summarizing, there was present total exclusion of the eighth nerve, accompanied by involvement of the fifth nerve and the cerebellum and signs of intracranial hypertension. The chronologic sequence of the complaints and the relationship of the findings left no doubt that we were dealing with a tumor of the acoustic nerve which was well into the neurologic stage. This was confirmed at operation. Histologic examination demonstrated the long cells in palisade arrangement typical of a neuroma of the acoustic nerve.

Case 2.—A man, aged 53, was admitted on the neurologic service with complaints of staggering gait for seven or eight years, dizziness, drooling and frontal headaches for the past few months. He had never had any tinnitus and was not conscious of deafness in either ear until examined.

The neurologic findings showed a peripheral facial paralysis and involvement of the fifth nerve on the left side, ataxia of both lower and upper extremities and slight papilledema. There was also a pulling over of the soft palate to the right on phonation, indicating paresis of the left ninth nerve. Cochlear examination of the right ear showed moderate impairment in hearing of a mixed type. On the left or involved side there was no hearing for the whispered voice, but tuning fork tests indicated the presence of a considerable remnant of hearing, including tones between *F*-90 and *a*-5 on the monochord. The *c*-256 fork used for the Rinne test could be heard for twenty-five seconds by air conduction and for thirty-five seconds by bone conduction, the latter being presumably borrowed from the other ear to some extent. The Weber test was not lateralized.

Vestibular examination showed spontaneous nystagmus essentially similar to that witnessed in the first case but only in the 1 degree position; the patient past pointed with the left hand to the left, and the Romberg sign was elicited mainly to the left and gait tendency to the left.

The caloric test gave the following results: Irrigation of the right ear after twenty-five seconds showed a 2 degree horizontal rotatory nystagmus to the left for ninety-six seconds; the patient past pointed to the right with the right hand and touched with the left.

Irrigation of the left ear (involved side) after fifty-one seconds showed a 2 degree horizontal rotatory nystagmus to the right for one hundred and five seconds. The patient past pointed correctly with each hand. With the patient's head back, a horizontal nystagmus could be seen in the midline in the proper direction after a douche was used in each ear.

Rotation showed a good horizontal nystagmus in the proper direction but somewhat diminished after rotation to the right. Vertigo was also in the proper direction but diminished. The falling test after rotation was in the correct direction. It was noted particularly in this patient that there was a complete lack of any susceptibility to stimulation.

In this case we were presented with a picture of an advanced tumor of the cerebellopontile angle, as indicated by the involvement of several cranial nerves, marked cerebellar signs and papilledema in which a definite amount of cochlear

function still persisted while vestibular function was only slightly impaired. It is true that remnants of hearing are not uncommon in fairly well advanced tumors of the acoustic nerve, but a combination of this together with a preservation of the vestibular nerve to the degree exhibited by this man was hardly conceivable except in an early stage. The diagnosis was therefore made of a tumor of the cerebellopontile angle not originating from the eighth nerve. The differentiation possesses a practical as well as a scientific implication since, as is well known, tumors of the acoustic nerve form a distinct entity with a benign course following removal, while other tumors in the vicinity clinically behave like a malignant growth. A specimen from the tumor of this patient was pronounced to be an oligodendroglioma by Dr. George Hassin of the neuropathology department. (The demonstration on the screen showed a loose tissue with relatively few cells and with no tendency whatever to any particular formation.)

It is to be noticed that neither of the patients exhibited the failure of the vertical canals on the opposite side to react to the cold caloric stimulus at 68 F., which has been featured rather prominently in American literature of recent years. This so-called "cerebellopontile angle syndrome," total loss of vestibular irritability on the homolateral side combined with loss of the reaction with the head, which was popularized by the late Dr. Lewis Fisher is, to judge from the reports of European observers, the exception rather than the rule in tumors of the eighth nerve and other growths in this region; in fact, hyperexcitability on the opposite side seems to have been found as frequently as hypo-excitability.

DISCUSSION

DR. ERIC OLDBERG: I wish to compliment Dr. Shapiro on having been able to differentiate these tumors by his caloric tests.

Clinically, they can also be differentiated in a high percentage of cases, provided a careful history is obtained. The reason for this is by virtue of the sites of origin of these two tumors of the cerebellopontile angle. Neuromas of the acoustic nerve, much the more common of the two, arise from the eighth nerve itself and, hence, give symptoms, usually tinnitus, long antedating all the other subsequent manifestations. As the tumor gradually enlarges, it begins to involve other cranial nerves, such as the seventh, the fifth and occasionally the ninth; eventually it attains sufficient size to encroach on the cerebellum, causing ataxia, and to produce internal hydrocephalus by pressure on the aqueduct of Sylvius with consequent headache, vomiting and choked disk.

Gliomas of the cerebellopontile angle, on the other hand, while giving exactly the same findings when fully developed, have an entirely different sequence of symptoms. They usually begin in the cerebellum, thereby causing cerebellar signs first. Later, as they extend down into the pons and surround the cranial nerves leaving that structure, they produce the other signs of tumor of the cerebellopontile angle; but symptoms of tumor of the eighth nerve, such as tinnitus, are usually late.

It is for the foregoing reason that caloric tests distinguish between the two types. With tumors of the acoustic nerve, the nerve is physiologically destroyed, whereas with gliomas of the cerebellopontile angle it may preserve some function because it is not primarily involved in the neoplasm.

As far as treatment is concerned, the neuromas of the acoustic nerve should all be operated on. They grow slowly, do not react to roentgenotherapy and, if not removed, eventually cause blindness from atrophy of the optic nerve, and death from pressure on the brain stem. The gliomas react well to roentgenotherapy, so that if they are diagnosed early, before signs of increased intracranial pressure have developed, they may be treated in this way. If, however, they are already showing signs of pressure in the form of choked disk, they should also be operated on, and sufficient tissue removed to at least unblock the aqueduct of Sylvius and permit of free circulation of cerebrospinal fluid.

The cases reported by Dr. Shapiro are typical in that the patients came to operation with the same group of neurologic findings, but that the chronology of symptoms and the caloric reactions were different.

Directory of Otolaryngologic Societies *

INTERNATIONAL

Second International Otolaryngological Congress

President: Prof. Dr. A. G. Tapia, Hermosilla, 2, Mod, Madrid, Spain.
Secretary: Prof. Dr. C. E. Benjamins, Verlengde, Heereweg 143, Gronigen, Holland.

FOREIGN

New Zealand British Medical Association, Ear, Nose and Throat Section

Chairman: Dr. James Hardie Neil, Auckland.
Secretary: Dr. W. Gillies Borrie, Auckland.

Royal Society of Medicine, Section of Laryngology

President: Dr. E. Musgrave Woodman, 22, Newhall St., Birmingham, England.
Secretary: Dr. A. Lowndes Yates, 33 Weymouth St., W. 1, London.

Royal Society of Medicine, Section of Otology

President: Dr. J. F. O'Malley, 6, Upper Wimpole St., W. 1, London.
Secretary: Dr. C. Gill Carey, 106, Harley St., W. 1, London.

NATIONAL

American Medical Association, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Harris P. Mosher, 828 Beacon St., Boston.
Secretary: Dr. John J. Shea, 1018 Madison Ave., Memphis, Tenn.

American Academy of Ophthalmology and Oto-Laryngology

President: Dr. B. R. Shurley, 62 W. Adams Ave., Detroit.
President-Elect: Dr. J. M. Wheeler, 30 W. 59th St., New York.
Executive Secretary: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Boston. Time: Sept. 18-22, 1933.

American Bronchoscopic Society

President: Dr. Richmond McKinney, 899 Madison Ave., Memphis, Tenn.
Secretary: Dr. Edwin McGinnis, 104 S. Michigan Ave., Chicago.

American Laryngological Association

President: Dr. George M. Coates, 1721 Pine St., Philadelphia.
Secretary: Dr. W. V. Mullin, 9204 Euclid Ave., Cleveland.

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* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

American Laryngological, Rhinological and Otological Society

President: Dr. Joseph C. Beck, 185 N. Wabash Ave., Chicago.
Secretary: Dr. R. L. Loughran, 33 E. Sixty-Third St., New York.

American Otological Society

President: Dr. John R. Page, 127 E. 62nd St., New York.
Secretary: Dr. Thomas J. Harris, 104 E. Fortieth St., New York.

SECTIONAL

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. R. H. Seeley, 9 Court St., Rutland, Vt.
Secretary-Treasurer: Dr. Arthur F. Holding, 142 Washington Ave., Albany.
Time: Third Wednesday of October, November, March, April, May and June.

New England Otological and Laryngological Society

President: Dr. Henry A. Barnes, 520 Commonwealth Ave., Boston.
Secretary-Treasurer: Dr. Lyman G. Richards, 319 Longwood Ave., Boston.

Pacific Coast Oto-Ophthalmological Society

President: Dr. Hans Barkan, 490 Post St., San Francisco.
Secretary-Treasurer: Dr. Frederick C. Cordes, 384 Post St., San Francisco.
Place: San Francisco. Time: June 28-30, 1933.

Puget Sound Academy of Ophthalmology and Oto-Laryngology

President: Dr. H. R. Secoy, 1802 Hewitt Ave., Everett, Wash.
Secretary-Treasurer: Dr. Francis A. Brugman, 806 Cobb Bldg., Seattle.
Time: 8 p. m., third Tuesday of each month, except July and August.

Saginaw Valley Academy of Ophthalmology and Otolaryngology

President: Dr. A. R. McKinney, 703 Second National Bank Bldg., Saginaw, Mich.
Secretary-Treasurer: Dr. W. K. Slack, 308 Eddy Bldg., Saginaw, Mich.
Place: Saginaw, Mich., or Bay City, Mich. Time: First Friday of each month except July and August.

Sioux Valley Eye and Ear Academy

President: Dr. John B. Gregg, 101 S. Main Ave., Sioux Falls, S. D.
Secretary-Treasurer: Dr. Frederick H. Roost, 420 Trimble Bldg., Sioux City, Iowa.

Southern Medical Association, Section on Eye, Ear, Nose and Throat

Chairman: Dr. W. D. Gill, 323 Medical Arts Bldg., San Antonio, Texas.
Secretary: Dr. W. A. Wagner, 200 Carondelet St., New Orleans.

Southwestern Michigan Triological Society

President: Dr. Carl Snapp, 604 Medical Arts Bldg., Grand Rapids, Mich.
Secretary: Dr. Lee O. Grant, 420 Medical Arts Bldg., Grand Rapids, Mich.
Time: Third Thursday of alternate months.

STATE

Colorado Oto-Laryngological Society

President: Dr. Harry L. Whitaker, 1612 Fremont St., Denver.
Secretary: Dr. Rex L. Murphy, Metropolitan Bldg., Denver.
Place: Assembly Room of Metropolitan Bldg. Time: First Saturday of each month from October to May.

Connecticut State Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. J. J. Cohane, 59 College St., New Haven.
Secretary-Treasurer: Dr. Walter L. Hogan, 750 Main St., Hartford.

Eye, Ear, Nose and Throat Club of Georgia

President: Dr. Thomas H. Hall, Jr., 617 Mulberry St., Macon.
Secretary-Treasurer: Dr. J. A. Smith, 700 Spring St., Macon.

Indiana Academy of Ophthalmology and Oto-Laryngology

President: Dr. Robert J. Masters, 805 Hume-Mansur Bldg., Indianapolis.
Secretary: Dr. J. Kent Leasure, 611 Hume-Mansur Bldg., Indianapolis.
Place: Indianapolis. Time: Dec. 13, 1933.

Iowa Academy of Ophthalmology and Oto-Laryngology

President: Dr. W. W. Pearson, 410 Sixth Ave., Des Moines.
Secretary-Treasurer: Dr. W. F. Boiler, 105 East Iowa Ave., Iowa City.

Michigan State Medical Society, Section of Ophthalmology and Otolaryngology

Chairman: Dr. Ray Connor, 28 W. Adams Ave., Detroit.
Secretary: Dr. Ralph B. Fast, 136 E. Michigan Ave., Kalamazoo.

Minnesota Academy of Ophthalmology and Oto-Laryngology

President: Dr. Harold J. Rothschild, 239 Lowry Bldg., St. Paul.
Secretary-Treasurer: Dr. W. E. Camp, Medical Arts Bldg., Minneapolis.
Time: Second Friday of each month, October to May.

Montana Academy of Oto-Ophthalmology

President: Dr. Lawrence G. Dunlap, 101 Main St., Anaconda.
Secretary: Dr. A. W. Morse, Phoenix Bldg., Butte.
Place: Anaconda. Time: July 11, 1933.

New Jersey State Medical Society, Section on Ophthalmology, Otology and Rhinolaryngology

Chairman: Dr. Charles H. Schlichter, 556 N. Broad St., Elizabeth.
Secretary: Dr. B. E. Failing, 31 Lincoln Park, Newark.

New York State Medical Society, Eye, Ear, Nose and Throat Section

Chairman: Dr. D. F. Gillette, 109 S. Warren St., Syracuse.
Secretary: Dr. Frank M. Sulzman, 1831 Fifth Ave., Troy.

NORTH DAKOTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Axel Oftedal, 55½ Broadway, Fargo.
Secretary-Treasurer: Dr. F. L. Wicks, 516 Fourth St., Valley City.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. A. B. Dykman, Medical Dental Bldg., Portland.
Secretary: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

RHODE ISLAND OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

President: Dr. Herman A. Winkler, 224 Thayer St., Providence.
Secretary: Dr. N. A. Bolotow, 108 Waterman St., Providence.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

SOUTH CAROLINA SOCIETY OF OTOLARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. Walter Bristow, Medical Arts Bldg., Columbia.
Secretary: Dr. J. W. Jervey, Jr., 101 Church St., Greenville.
Place: Columbia. Time: October, 1933.

TENNESSEE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. C. D. Blassingame, 899 Madison Ave., Memphis.
Secretary: Dr. Ralph O. Rychener, 130 Madison Ave., Memphis.

TEXAS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. A. F. Clark, 388 Pearl St., Beaumont, Texas.
Secretary: Dr. A. F. Clark, 1034 Nix Bldg., San Antonio.
Place: Dallas. Time: Dec. 1-2, 1933.

UTAH OPHTHALMOLOGICAL SOCIETY

President: Dr. F. M. McHugh, 17 Exchange Pl., Salt Lake City.
Secretary-Treasurer: Dr. W. Leroy Smith, Erza Thompson Bldg., Salt Lake City.

VIRGINIA SOCIETY OF OTOLARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. Frank P. Smart, 142 W. York St., Norfolk.
Secretary-Treasurer: Dr. Henry Grant Preston, Professional Bldg., Harrisonburg.

WEST VIRGINIA STATE MEDICAL ASSOCIATION, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. O. H. Bobbitt, Medical Arts Bldg., Charleston.
Secretary: Dr. Harry V. Thomas, 95 Fairmont Ave., Fairmont.

LOCAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Elbert A. Curtis, 65 Central St., Newark.
Secretary: Dr. A. Russell Sherman, 671 Broad St., Newark.
Place: 91 Lincoln Park South. Time: 8:45 p. m., second Monday of each month.

ATLANTA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. G. E. Clay, 384 Peachtree St., N. E., Atlanta, Ga.
Secretary: Dr. William O. Martin, Jr., 412 Medical Arts Bldg., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St., N. E. Time: 8 p. m., last Thursday of each month.

Baltimore City Medical Society, Section on Otology and Laryngology

President: Dr. James J. Chisolm, Medical Arts Bldg., Baltimore.
Secretary: Dr. Louis Diener, 2449 Eutaw Pl., Baltimore.
Place: 1211 Cathedral St. Time: 8:30 p. m., last Friday of each month.

Buffalo Oto-Laryngological Society

President: Dr. Charles B. Handel, 131 Linwood Ave., Buffalo.
Secretary-Treasurer: Dr. Carl G. Frost, 37 Allen St., Buffalo.
Place: Buffalo Club. Time: First Tuesday of each month from September to May, inclusive.

Chicago Laryngological and Otological Society

President: Dr. Charles Younger, 25 E. Washington St., Chicago.
Secretary-Treasurer: Dr. Austin A. Hayden, 25 E. Washington St., Chicago.
Place: Medical and Dental Arts Bldg., 185 N. Wabash Ave. Time: 6 p. m., first Monday of each month from October to May.

Cincinnati Oto-Laryngological Society

President: Dr. Henry M. Goodyear, The Doctors' Bldg., Cincinnati.
Secretary-Treasurer: Dr. Samuel Seltz, Provident Bank Bldg., Cincinnati.
Place: University Club. Time: 6:30 p. m., third Tuesday of each month from October to May.

Cleveland Academy of Medicine, Section of Ophthalmology and Otolaryngology

Chairman: Dr. Justin M. Waugh, Cleveland Clinic, Cleveland.
Secretary: Dr. James T. Collins, 1105 Medical Arts Bldg., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

College of Physicians of Philadelphia, Section on Otolaryngology

Chairman: Dr. Ralph Butler, 2007 Chestnut St., Philadelphia.
Clerk: Dr. Karl M. Houser, 1826 Pine St., Philadelphia.
Place: College of Physicians. Time: 8:30 p. m., third Wednesday of each month from October to June.

Columbus Ophthalmological and Otolaryngological Society

Chairman: Dr. John B. Alcorn, 167 E. State St., Columbus, Ohio.
Secretary-Treasurer: Dr. Edward W. Harris, 681 E. Broad St., Columbus, Ohio.
Time: First Monday of each month.

Dallas Academy of Ophthalmology and Otolaryngology

President: Dr. O. M. Marchman, 1717 Pacific Ave., Dallas, Texas.
Recording Secretary: Dr. W. Mood Knowles, 1717 Pacific Ave., Dallas, Texas.
Corresponding Secretary: Dr. J. M. Potts, 1717 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Des Moines Academy of Ophthalmology and Otolaryngology

President: Dr. C. F. Howland, 604 Locust St., Des Moines, Iowa.
Secretary-Treasurer: Dr. Grace Doane, 614 Bankers Trust Bldg., Des Moines, Ia.
Time: Third Monday of each month, September to May, inclusive.

Detroit Oto-Laryngological Society

President: Dr. R. Lee Laird, 513 David Whitney Bldg., Detroit.
Secretary-Treasurer: Dr. J. M. Sutherland, 8-257 General Motors Bldg., Detroit.
Place: Maccabees Bldg. Time: Third Wednesday evening of each month from October to May.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. R. H. Needham, 1304 Medical Arts Bldg., Fort Worth, Texas.
Secretary: Dr. Van D. Rathgeber, 1011 Medical Arts Bldg., Fort Worth, Texas.
Place: University Club. Time: 6.30 p. m., first Friday of each month except July and August.

Grand Rapids Eye, Ear, Nose and Throat Society

President: Dr. Carl F. Snapp, Medical Arts Bldg., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Walter W. Oliver, 26 Sheldon Ave., Grand Rapids, Mich.
Place: Various local hospitals. Time: 6:30 p. m., third Thursday of alternating months, September to May.

Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. J. F. Gamble, 201 Main St., Houston, Tex.
Secretary: Dr. B. F. Payne, 1304 Walker Ave., Houston, Tex.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

Indianapolis Ophthalmological and Oto-Laryngological Society

President: Dr. John W. Carmack, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: Indianapolis Athletic Club. Time: 6:30 p. m., second Thursday of each month from October to May.

Kansas City Society of Ophthalmology and Otolaryngology

President: Dr. Albert N. Lemoine, 906 Grand Ave., Kansas City, Mo.
Secretary: Dr. Harold B. Hedrick, 618 Professional Bldg., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

President: Dr. Clifford S. Losey, 219 E. 10th St., Long Beach, Calif.
Secretary-Treasurer: Dr. Kenneth C. Brandenburg, 707 Security Bldg., Long Beach, Calif.
Place: Professional Bldg. Time: Last Wednesday of each month from September to June.

Los Angeles Society of Ophthalmology and Otolaryngology

President: Dr. D. E. Godwin, 910 Security Bldg., Long Beach, Calif.
Secretary-Treasurer: Dr. F. H. Brandt, 1052 W. Sixth St., Los Angeles.
Place: Los Angeles County Medical Association, 1925 Wilshire Blvd. Time: 8 p. m., fourth Monday of each month, September to May, inclusive.

Louisville Eye, Ear, Nose and Throat Society

President: Dr. Karl N. Victor, Breslin Bldg., Louisville, Ky.
Secretary-Treasurer: Dr. J. S. Bumgardner, Heyburn Bldg., Louisville, Ky.
Place: French Village. Time: 6:30 p. m., second Thursday of each month, September through May.

Medical Society of the County of Kings, Section on Otolaryngology

Chairman: Dr. Charles R. Weeth, 37 Remsen St., Brooklyn.
Secretary-Treasurer: Dr. Oscar Rodin, 893 Eastern Parkway, Brooklyn.
Place: Library Bldg., County Society, 1313 Bedford St., Brooklyn.
Time: 9 p. m., second Wednesday of January, March, May and November.

Medical Society of the District of Columbia, Section on Ophthalmology and Oto-Laryngology

Chairman: Dr. Albert P. Tibbets, 1801 I St., Washington.
Secretary: Dr. G. Victor Simpson, 1710 Rhode Island Ave., Washington.
Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month.

Memphis Society of Ophthalmology and Oto-Laryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. R. O. Rychener, Exchange Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

Milwaukee Oto-Ophthalmic Society

President: Dr. Samuel G. Higgins, 324 E. Wisconsin Ave., Milwaukee.
Secretary-Treasurer: Dr. O. P. Schoofs, 324 E. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6:30 p. m., third Tuesday of each month.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. Eugene Orr, 706 Church St., Nashville.
Secretary-Treasurer: Dr. H. C. Smith, Medical Bldg., Nashville.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month, October to June.

New Orleans Ophthalmological and Otolaryngological Society

President: Dr. M. F. Meyer, Canal Bank Bldg., New Orleans.
Secretary-Treasurer: Dr. R. H. Fisher, Chess and Checkers Club, New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: January to June and October to January—third Thursday of each month.

New York Academy of Medicine, Section of Otolaryngology

Chairman: Dr. Samuel J. Kopetzky, 51 W. 73rd St., New York.
Secretary: Dr. Wallace Morrison, 39 E. 50th St., New York.
Place: 103d St. and 5th Ave. Time: 8:30 p. m., third Wednesday of each month from October to May.

Omaha and Council Bluffs Ophthalmological and Otolaryngological Society

President: Dr. L. G. Howard, 532, 1st Ave., Council Bluffs, Iowa.
Secretary-Treasurer: Dr. W. A. Cassidy, 1620 Medical Arts Bldg., Omaha.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: Dinner, 6 p. m. Program, 7 p. m., third Wednesday of each month from October to May.

Philadelphia Laryngological Society

President: Dr. Louis H. Clerf, 128 S. Tenth St., Philadelphia.
Secretary: Dr. Herman B. Cohen, 1301 Spruce St., Philadelphia.
Place: College of Physicians. Time: 8:30 p. m., first Tuesday of each month from October to June.

Pittsburgh Otological Society

Chairman: Dr. George B. Jobson, 1238 Liberty St., Franklin, Pa.
Secretary-Treasurer: Dr. John R. Simpson, Medical Arts Bldg., Pittsburgh.
Place: Pittsburgh Academy of Medicine, Craig St., Pittsburgh.

Richmond Ophthalmological and Oto-laryngological Society

President: Dr. R. W. Vaughan, Medical Arts Bldg., Richmond, Va.
Secretary: Dr. William R. Weisiger, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

Rochester Eye, Ear, Nose and Throat Society

Chairman: Dr. E. S. Amsler, 53 S. Fitzhugh St., Rochester, N. Y.
Secretary-Treasurer: Dr. E. W. O'Brien, 5 N. Goodman St., Rochester, N. Y.
Place: Rochester Medical Association, 13 Prince St. Time: 8 p. m., third Monday of each month from October to May.

St. Louis Ear, Nose and Throat Club

Acting President: Senior member of the executive committee: Dr. L. W. Dean, Washington University School of Medicine, St. Louis.
Secretary: Dr. B. J. McMahon, 813 Missouri Bldg., St. Louis.
Place: University Club Bldg. Time: Third Wednesday in February, April, October and December.

San Antonio Ophthalmolo-Oto-Laryngological Society

President: Dr. Robert E. Parrish, 712 Medical Arts Bldg., San Antonio, Texas.
Secretary-Treasurer: Dr. E. King Gill, 323 Medical Arts Bldg., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Robert Carson Martin, 384 Post St., San Francisco.
Secretary: Dr. George N. Hosford, 490 Post St., San Francisco.
Place: Society's Building, 2180 Washington St., San Francisco. Time: Third Tuesday of every month except May, June, July and December.

Shreveport Eye, Ear, Nose and Throat Society

President: Dr. T. D. Boaz, Medical Arts Bldg., Shreveport, La.
Secretary-Treasurer: Dr. W. L. Atkins, 940 Margaret Pl., Shreveport, La.
Place: 1240 Texas Ave. Time: 7:30 p. m., first Monday of each month from October to June.

Spokane Academy of Ophthalmology and Otolaryngology

President: Dr. C. A. Veasey, Jr., Paulsen Medical and Dental Bldg., Spokane, Wash.
Secretary: Dr. J. L. Walter, Paulsen Medical and Dental Bldg., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. W. G. Dickinson, 109 S. Warren St., Syracuse, N. Y.
Secretary-Treasurer: Dr. Harold H. Joy, 109 S. Warren St., Syracuse, N. Y.
Place: University Club. Time: Second Friday of each month except June, July and August.

Tulsa Academy of Ophthalmology and Otolaryngology

President: Dr. Roy W. Dunlap, 108 W. 6th St., Tulsa, Okla.
Secretary-Treasurer: Dr. Marvin D. Henley, 108 W. 6th St., Tulsa, Okla.
Place: Medical Arts Bldg. Time: Monthly.

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